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RENAL DWARFISM.[1]

By KEMPSON MADDOX, M.D. (Sydney), M.R.C.P. (London),

Honorary Assistant Physician and Student Supervisor, Royal Prince Alfred Hospital, Sydney.

RENAL dwarfism, renal infantilism, renal rickets, renal nanism or renal pseudo-rickets, is defined by Barber[1] as "a condition of children marked by stunted development, often associated with bone deformities of 'late rickets' type and symptoms of a uræmic character, due to a severe chronic renal insufficiency, frequently of congenital origin." It is one form of "late rickets" or the *"rhachitis tarda"* of older writers, and undoubtedly some such cases have occasionally been of renal origin. Thus Ashcroft[2] found that 33% of 24 children with this diagnosis showed definite renal insufficiency.

[1] Abstract of a thesis submitted for the degree of Doctor of Medicine, University of Sydney, 1930.

Early descriptions of the condition appeared in the writings of Goodhart,[3] Barlow[4] and Cheadle[5] in the latter part of the nineteenth century, and Clement Lucas[6] in 1883 observed that "the phenomena of late rickets and albuminuria are too frequently connected to be matters of chance". The subject was revived by contributions of single case reports by Sequeira,[7] Guthrie,[8] Milligan[9] and Sawyer[10] at the beginning of the present century. Since then several Continental physicians, and Morley Fletcher,[11] Miller,[12] Parsons,[13] Paterson[14] and, in particular, Barber,[1] have further elucidated the subject.

British authors have contributed 80% of the literature. Hunt[15] in 1927 was able to collect 53 cases, including one of his own. I have details of 84 cases drawn from literature in London, Melbourne and Sydney, inclusive of three cases which have come under my personal observation and are now described.

Case I.—A.P., a female, aged nineteen years, first came under my care with her condition undiagnosed during the terminal phase of her illness. Both parents were rather undersized (the father was just 150 centimetres or five feet high), but healthy. There was no other family history of dwarfism, renal disease, syphilis or tuberculosis. There had been no trouble during the pregnancy or confinement, and one other healthy child was born subsequent to the patient. The patient had suffered from uncomplicated bouts of measles and whooping cough before the age of two years and had never contracted diphtheria or scarlet fever. Until eleven years of age the child seemed to be quite normal, both mentally and physically, and received an appropriate education. She then began to "go off her feet", her bones began to bend, and three years later she was unable to walk at all. Though she had frequent falls, she was never known to have sustained any fractures. Her mentality remained that of a child of eleven years; she could knit and read simple books. At this point her parents first noticed her great thirst. She also had some defect in vision, with much headache and general weakness. For a few years she was wheeled about in a chair, her health alternating between moderately good and poor. Her skin became sallow and yellow looking. Her appetite on the whole was good, but the bowels were continually constipated. She would micturate frequently and during the night; the quantity of urine was not observed to be particularly great. She never at any time had vomiting, convulsions or seizures resembling tetanic spasms. For the last twelve months she had been wholly confined to bed and lately had developed some pressure sores over bony prominences, including a troublesome one over the sacrum; her limbs and spine had become more bent and distorted than ever.

There had been no attempt at restriction of her diet at any period. Quite suddenly her urinary output diminished very much; she became drowsy, with a furred tongue, and had a urinous odour to her breath. The pulse rate commenced to rise progressively. She now began to be affected by sudden and acute attacks of laryngeal stridor, accompanied by marked recession in the lower part of the thorax. This made her very terrified, for the attacks were excited by the slightest extraneous stimulus, for example, by anyone walking into the room. Chvostek's test gave an extremely positive reaction, a marked facial contortion resulting. Trousseau's sign was not present, though the hands were kept continually clenched in the tetanic posture, her feet were less definitely clenched. She gradually sank into a deepening coma, in which the *laryngismus stridulus* played no part, until her death six days after the onset of her terminal illness.

General physical examination four days before death showed that she was extremely dwarfed and deformed, measuring only 99 centimetres (three and a quarter feet) in length and weighing 22 kilograms (three and a half stone). She had an earthy yellow complexion with hollow cheeks and a dry, harsh skin, though her eyes appeared very bright and observant, her facies resembling that of an old woman. Pubic hair and breast enlargement were present to the degree usually shown about six months before puberty. Her teeth were remarkably good and showed no signs of deficient calcification. The tongue was coated. The mucous membranes showed great anæmia. Fauces and neck were clear and the thyreoid appeared normal. Her thorax was laterally compressed, with a tendency to pigeon breast, and showed very marked "beading" (see Figure I). Respiration was abdominal in character, and her chest was free from abnormal physical signs apart from some fine basal crepitations. Her heart appeared somewhat enlarged to the left, but this was uncertain owing to the deformed condition of the thorax; and the radial vessels were hard and thickened. Her systolic blood pressure was 140, and her diastolic pressure was 100 millimetres of mercury. The abdomen appeared full, with hard scybala palpable along the line of the colon. The kidneys, liver and spleen were not palpated. There was generalized muscular wasting, which reached a very marked degree, except in the upper limbs, with hypotonia and weakness. Her cranial nerves were unaffected beyond some irritability of the facial nerve. Deep reflexes were present with the exception of the knee jerks. The fundi were the seat of definite albuminuric retinitis.

Bony deformity was extreme. The skull was normal in size, with closed fontanelle, and no craniotabes could be detected; definite kyphosis was evident in the spine. The pelvis was diminutive. All the limbs were extremely deformed, so that every long bone was involved, being twisted and curved in all directions, besides being actually slightly pliable in the hands. The lower limbs were most affected, being permanently contracted in adduction and flexion at the hip and knee. The femora were almost semicircular and projected beneath the skin of the anterior aspect of the thighs.

Figure I.

Autopsy illustration of Case I. Note the adult facies, enlarged wrists and costo-chondral junctions, contortion of the lower limbs and pyogenic invasion of the skin. The site of the old fracture in the femur is plainly visible. Disproportion in size between feet and legs, while it exists, is exaggerated by this photograph.

The epiphyses of the wrists, knees and ankles were especially enlarged. The tibiæ were actually bent at their lower ends almost to a right angle, closely resembling the illustration of one of Parsons's cases, recently reported, and apparently due to the weight of the limb on the bed. The disproportion between the feet and the legs was very striking. There was no œdema to be found anywhere, and a bed sore about five centimetres across was situated over the lower half of the sacrum.

Urinary examination revealed an unmeasured polyuria until four days before death, with a small variable quantity of albumin and some odd hyaline casts. Specific gravity averaged 1·004 to 1·010. Subsequently the urine diminished very much in amount, and became almost solid on boiling.

Only one collection of blood could be taken, and was sent to the Liverpool Medical School Laboratory, which reported as follows: Urea, 535 milligrammes per 100 cubic centimetres of plasma; inorganic calcium, 4·1 milligrammes per 100 cubic centimetres of plasma; inorganic phosphate, 7·48 milligrammes per 100 cubic centimetres of plasma.

Findings at autopsy were as follows: The brain appeared moist and rather congested, with flattened convolutions, while the cerebral vessels showed some atheroma, but otherwise appeared fairly normal. The thyreoid was small, but healthy. The lungs were normal apart from some congestion along their posterior borders. The left ventricle was somewhat thicker than usual, but the muscle, valves, coronary vessels and aorta displayed no abnormality. Liver, spleen, intestines and mesenteric vessels were free from change, but the colon was loaded and the pancreas harder than normal. Suprarenals appeared unaffected and were as large as the kidneys themselves. Each kidney was extremely shrivelled, with a papillated granular surface, and was approximately 5·0 by 4·5 by 2·0 centimetres in size. Together they weighed 42 grammes. One or two small cysts, about three millimetres in diameter, appeared on the surface. On section the cortex appeared as a narrow, tough rind. The pelvis was hydronephrotic and had encroached on the medulla in one kidney, and the ureter on this side was also definitely dilated. The pelvic fat was not increased and the vessels gaped prominently (Figures II and IIa).

The bladder was large and hypertrophied, as if accustomed to dealing with large quantities of urine. The uterus and tubes were hypoplastic and the ovaries were

unscarred. There was no obstruction to the passage of a probe through the urethra or lower half of the ureter. The long bones were extraordinarily distorted, being actually plastic (Figure III). An old poorly healed fracture appeared in the middle third of the right femur (Figure IIIA). The skull appeared to cut easily, but no other change could be detected from either aspect. The thorax was especially soft, with great enlargement of the costo-chondral junctions, less on the inner than the outer side. On section, all the long bones were thin and atrophic, with an attempt at thickening along their concave borders and a sharp increase in breadth at the epiphyseal line. The changes were most plainly visible at the lower extremity of the femur or upper end of the humerus. The marrow was pale and fatty, the cortex thick and soft. The widened epiphyseal line was fairly regular along the distal border, but proximally was completely broken up and connected with outlying islands of cartilage in the metaphysis, which appeared quite vascular (Figure IV). Nothing further could be made out by the naked eye. In none of the epiphyses was there any true bony collapse or dislocation.

FIGURE II.

Kidney from Case I (twice natural size). Note the extreme atrophy associated with almost complete disappearance of the parenchyma and a relative degree of hydronephrosis and hydroureter. The cause of the latter was obscure, but may be partly compensatory or related to the large amount of urine passed.

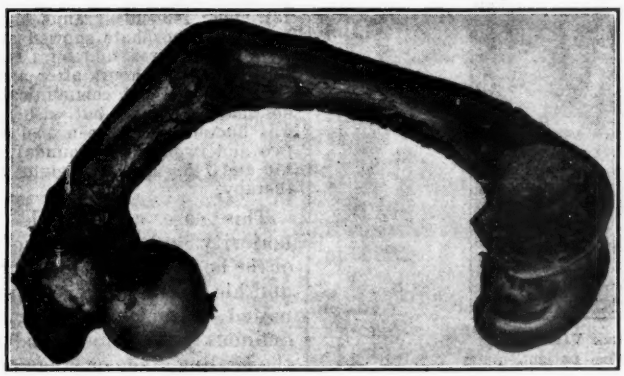

FIGURE IIIA.

Femur from Case I. Note the characteristic serration of the epiphyseal lines in the lower end of the bone.

Microscopical appearances were also very typical. Sections of the kidney were stained by the iron hæmatoxylin and Van Gieson, hæmatoxylin and eosin, iron hæmatoxylin and picro-indigo carmine techniques. They showed the capsule to be grossly thickened and consisting of dense old fibrous tissue connecting in the depressions with equally dense radiating scars in which were a few scattered remains of dead atrophied tubules. Between these, and in close contact with each other, were extremely large, hypertrophied and active-looking glomeruli, showing, however, hyalinization, and in places adherent to the capsule (Figures V and VA). Dead glomeruli were barely recognizable as such from the dilated *débris*-filled tubules. Only a certain number of tubules of all sizes remained, and of these none were normal. In all, complete desquamation of epithelium had occurred, and each was ensheathed with a varying amount of fibrous tissue. The vessels were only moderately thickened, and small round cells abounded in groups throughout the cortex. Owing to the encroachment of the pelvis on the medulla, this was very limited in extent and distinguishable from the cortex only by the absence of glomeruli.

FIGURE IIA.

Kidney from Case I (twice natural size). Note the extreme atrophy associated with almost complete disappearance of the parenchyma and a relative degree of hydronephrosis and hydroureter. The cause of the latter was obscure, but may be partly compensatory or related to the large amount of urine passed.

Sections were also made, without any necessity for decalcification, from the lower end of the femur and stained by the above methods. They showed a thickened periosteum, poorly formed bone with slender trabeculæ, large Haversian canals, and abundant fibro-fatty marrow. Strands of well-formed fibrous tissue marked the irregular junction of the trabeculæ of the shaft with the cartilage, which consisted of a completely irregular arrangement of degenerate cells, similar to those forming the cartilaginous nodules in the shaft. Osteoblasts were scanty and osteoclasts absent. The general impression was one of active absorption and cartilaginous invasion, like an endochondroma.

Radiographs, taken after death, showed the bones to be extremely decalcified and the shafts remarkably stippled (Figure VI), corresponding also in epiphyseal appearances to the "woolly" type of Parsons (*vide infra*).

This is the most extreme, but not the most prolonged example of the disease to be found in the literature, and it was unfortunate that the patient was under observation for so short a period. It is noteworthy that nothing apparently happened at eleven years of age, except perhaps the beginnings of puberty, to start her on the downward grade. After her confinement to bed for five years, some unstable kind of renal balance was apparently established, which perhaps was finally upset by the toxæmia from bed sores, a loaded bowel and unsuitable diet.

Figure VI.

Humerus and radius from Case I. Note the characteristic serration of the epiphyseal lines in the upper end of the humerus. The splaying of the lower end of the radius is very striking.

Case II.[1]—V.C.M., a male, aged nine years, was admitted to hospital in August, 1928, complaining of dwarfism and fits. He was the elder of two sons of healthy parents. His mother's pregnancy and confinement were without incident. She had had no miscarriages. The patient's health had been good, except for "stomatitis" and pains in his legs at three years. The younger son was seven years old, and sturdy, weighing 41 kilograms (six and a half stone). The patient was breast fed till three months old and was then given cow's milk. He began to have occasional fits at two years of age. These were preceded by headache and drowsiness, and began with vomiting, after which he became "blue, stiff and unconscious". Up to the age of five years he had several seizures a day, but since then they had averaged one about every six weeks. Well marked polyuria and polydipsia had been present for years. Knock-knees had appeared during the last ten months. His appetite was good with an ordinary diet, but the bowels were costive. The lad appeared pale and very thin, small, but intelligent, with an earthy complexion and hollow cheeks. He was 87·5 centimetres (thirty-five inches) in height and weighed only 10·9 kilograms (twenty-four pounds). His tongue was clean and his throat healthy. The lungs were clear and the heart's action forcible, but with no noticeable increase in its usual boundaries at this age. The radial vessels, however, were thick and hypertrophied. Blood pressure at this time showed readings of 120 millimetres of mercury, systolic, and 80 millimetres, diastolic. The abdomen was clear and the kidneys could not be felt. The optic disks showed thickening of the retinal vessels, but were otherwise clear. Bone changes were notable, as though some "beading" could be made out, together with a Harrison's sulcus and flattened thorax, there was no distinct enlargement of epiphyses. The femora and tibiæ, however, were very much curved in both directions, resulting in a marked *genu valgum*. The urine was acid, with a low specific gravity (1·008). A cloud of albumin was present intermittently. Blood and casts were absent. Urinary urea amounted to 6%. The urinary chloride was normal, and acetone was found only once. Radiograms showed curving of the long bones, but no great rarefaction. There was some irregularity and cupping of the epiphyseal line. The epiphyses themselves, however, appeared normal in size and position. There appeared to be definite sclerosis on the shaft side of the epiphyseal line, as if more or less unsuccessful attempts had been made at healing.

Figure VII.

Case III. The patient, aged thirteen, compared with a normal child of eleven years. Note the *genu valgum* and the contrast in expression.

Blood chemistry was as follows: Blood urea readings in six weeks showed a consecutive increase thus: 108, 121, 140, 142 milligrammes per 100 cubic centimetres. Inorganic phosphate showed at 3·82 milligrammes per 100 cubic centimetres, and total calcium at 10·4 milligrammes. Blood fat three hours after a meal registered 0·575% fatty acids. When last communicated with (September, 1928) he was fairly well, but with a poor appetite. His weight was fluctuating between 10·0 and 10·9 kilograms (twenty-two and twenty-four pounds), and his pulse rate between 110 and 115. He lost weight after a course of ultra-violet therapy.

This case corresponds fairly closely with the majority, but at the time of observation the state of the bones as revealed by radiograms was improved and his blood phosphate and blood calcium approximated to normal limits; so that, in spite of an ominous rise in his blood urea, he was fairly into the healing stage or stage of remission. His frequent fits, in the light of the associated uræmic symptoms, must be regarded as uræmic rather than epileptic phenomena. There are unfortunately no photographs or radiograms of this case available for reproduction.

Case III.—I.C., a female, aged thirteen years, was admitted to hospital on February 4, 1930, with the diagnosis of acute leuchæmia. She was the third child of ten and the daughter of a Zulu half-caste. The remaining children were all healthy and, as far as could be dis-

[1] I am indebted to Dr. D. H. Paterson for permission to make some notes on this case, which was under his care at Great Ormond Street Hospital. The biochemical changes were observed by Dr. Payne, Biochemist to the Hospital.

covered, there was no history of renal disease in the family. During the pregnancy the mother suffered from "kidney disease", further details of which will be given later. The mother explained that the child was delicate as a baby, and had to be weaned early. At eight months she became very thin and thirsty. She had been thirsty ever since and has always wet the bed a good deal. She would not take any solid food until she was three years old, but had not had any vomiting fits. She had an attack of varicella at two years and of mumps at seven years. Her tonsils and adenoids were removed at nine years without incident. There was a very doubtful history of diphtheria and none at all of scarlet fever.[1]

Her knock-knees have been under notice for eighteen months. She has of late complained frequently of headache. Usually her appetite is good, but her bowels are always obstinate. Eleven days before her admission she had pain on the left side of her chest with some cough, became noticeably thinner and paler, drowsy and difficult to rouse. When first seen by me she had quite recovered from this attack, which was certainly due to an infection and not wholly uræmic. She was markedly dwarfed, being only 125 centimetres (four feet two inches) in height—that of a normal child of nine years. She weighed 25 kilograms (four stone and half a pound) (Figure VII). Her complexion was pale and her expression mature and intelligent. A broad, thick nose was reminiscent of her ancestry. There was a spotty pigmentation over the whole of the middle third of her face. Her lips and mucous membranes were quite pale, but not pigmented. There were no signs of puberty. Her hair and skin appeared to be of quite normal texture, and the musculature of moderately good tone and development. Bone changes were most pronounced in the legs, with fully 15 centimetres (six inches) of separation of the heels, due to *genu valgum*. The wrists showed slight enlargement; the knees were more definitely enlarged. There was a distinctly large "rosary" and a Harrison's sulcus in the chest wall. The fundi were clear. There was no apparent cardio-vascular hypertrophy. The blood pressure was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic. The abdomen appeared very full and there was suggestive evidence of some free fluid. The liver was just palpable, the spleen not at all.

A blood count revealed a moderate secondary anæmia and negatived the diagnosis of leuchæmia. Blood platelets were observed to be very abundant. The urine was acid, specific gravity 1·006, with an elusive cloud of albumin and a few odd casts. Wassermann, Kahn and von Pirquet tests all gave no reactions. The epinephrine test for Addison's disease definitely gave no reaction.

Biochemical estimations of the urea, calcium and phosphorus of the blood were performed as follows: (*a*) Blood urea, 31 milligrammes per 100 cubic centimetres; four days later, 62 milligrammes per 100 cubic centimetres. (*b*) Inorganic phosphorus, 7·5 milligrammes per 100 cubic centimetres. (*c*) Inorganic calcium, 6·6 milligrammes per 100 cubic centimetres.

Using McLean's urea concentration test, a concentration of 1·9% urea was all that could be obtained after two hours.

Radiograms proved diagnostic both in the skull and at the ankles (Figures VIII and IX) and showed characteristic serration of the epiphyseal line, with linear markings and sclerosed zones marking previous attempts at healing. There was no collapse or bending of the shaft, though the bones showed an advanced grade of generalized rarefaction. In the skull were small rounded areas of osteosclerosis towards the base, with a wool-like dense thickening of the posterior part of the vault (Figure IX). She proved active and useful while in hospital, with quite a normal intelligence. She received no special treatment and her condition seemed stationary.

[1] This patient was admitted to the Royal Alexandra Hospital for Children on February 4, 1930, under the care of Dr. McIntosh. He showed her at a British Medical Association clinical meeting and has kindly allowed me to see her and make use of my notes for the present purpose. Dr. Sear was also good enough to permit me to reproduce the radiograms (Figure IX).

Clinical Features.

Age of Onset.

The average age of the patients collected from the literature on their first coming under observation was seven years. Lathrop, reviewing fourteen cases, places this average at twelve and a half years. The first appearance of *genu valgum* is commonly between twelve and fourteen years of age, but Parsons,[13] Paterson[14] and Brockman[16] have described well marked bony changes under sixteen months of age. Among 72 patients there are 39 males and 33 females.

Symptoms.

Symptoms usually appear in the following order.

1. Polydipsia is noted in 40 of 72 cases and is specially prominent under three years of age, when the child tends to cling to the all-liquid type of diet.

2. Polyuria becomes progressively greater.

3. Development is retarded. The slow epiphyseal growth is complemented by the reduction in actual height caused by the severe degree of *genu valgum*. Some of the most extreme examples of dwarfism are due to this cause. In 17 of 84 cases dwarfism occurs in which no mention is made of any clinical rickets. This type of case in almost every instance goes back to infancy. In general there is much more dwarfism than can be accounted for by clinical changes in the bones themselves, for example, by dislocation, *genu valgum* and bending. There is thus an actual slowing up of the process of bone deposition, as if the body were adapting its bulk to the diminished renal reserve. Intelligence is very fair, being proportional to the extent of the toxæmia and to the degree of interference with education caused by invalidism. The facies and expression are often surprisingly mature.

4. Pain in the knees was noted in 5% of cases, and probably arises (in the severe types of deformity) from the abnormal ligamentous strain.

5. Symptoms of uræmia, such as anorexia, constipation, vomiting and convulsions, are usually present in varying degree, and, with the exception of dyspnœa and oliguria, may be present for many years before death. Convulsions have not necessarily been associated with a raised blood pressure.

6. Infantilism in some degree, but never complete, is usually apparent in cases beyond the age of puberty.

7. Pallor, or a pale, dry, wrinkled skin, with absence of sweating, is common in any case of azotæmic nephritis. The blood count shows a secondary anæmia.

8. Pigmentation appears to be more common than is usual with nephritic adults. It is patchy and blotchy in distribution, most evident on the face, and was noted in seventeen of the cases under review.

9. Cardio-vascular hypertrophy is a very variable occurrence, and seems to lag behind the other features of the syndrome. It seems to be more common in the cases at puberty and later, but Case

II was remarkable in showing considerable arterial hypertrophy without a proportional rise of blood pressure. Of thirty cases the systolic pressure was raised in nine to a high degree and in three to a slight degree, the average of the first group being 188 millimetres of mercury. Interference with bone growth appears to be a result of the nephritis quite distinct from the production of a high blood pressure.

10. Fundal changes, when present, are typical of adult renal retinitis, though the children have apparently not suffered much ocular inconvenience.

The Bone Deformities.

The bone deformities are as follows:

1. *Genu Valgum.* This important sign is best seen in the older children, or in younger children only after walking commences. It is frequently exaggerated by some synchronous curving of the femora and well marked epiphyseal enlargement. To the orthopædist it is the type of knock-knee which is beyond hope of cure by splinting and is in need of an osteotomy, so that numbers of these patients have been discovered in surgical departments. There is frequently a separation of 10 to 25 centimetres (four to ten inches) between the heels. It may appear quite suddenly. Of any three cases of adolescent knock-knee at lease two will probably be due to renal dwarfism (Ashcroft[2]).

2. *Enlargement of the Epiphyses.* Enlargement of the epiphyses is best looked for at the wrists and ankles, and is frequently very pronounced (Figure I).

3. *Changes in the Skull.* There is frequently no clinical alteration in the skull, but the fontanelle may remain open and bossing has rarely been noted.

4. *Changes in the Thorax.* Changes in the thorax are of the same order as occur in nutritional rickets, for example, enlargement of the costo-chondral junctions, Harrison's sulcus and pigeon-breast.

5. *Malacia of the Long Bones.*—Malacia of the long bones is present in only the very advanced type of case (Figure I). Fractures occur, but are not common, and their unions are weak. Some are really examples of paraepiphyseal collapse.

Changes in the Body Fluids.

Urine. The urine shows all the accepted features of an established chronic nephritis, including a constant low fixation of the specific gravity, with an increase of the night urine, a variable and elusive cloud of albumin, odd red cells, hyaline and granular casts.

The Blood. Blood urea, total non-protein nitrogen, uric acid and creatinine, all show a progressive rise as death approaches, the highest figure recorded being 720 milligrammes of urea per 100 cubic centimetres. Chlorides were retained and plasma cholesterol was not raised in Cockayne's[17] case. Lipæmia may occur and be extreme.

In regard to blood phosphorus and blood calcium, of twelve available cases three show a normal reading of blood phosphate; in the remainder these readings are all raised to a varying degree. A very high phosphorus reading (14 milligrammes *per centum* or more) is apparently found only within a short time of death, but sudden falls can occur, while no case is reported as showing a constantly subnormal phosphate level. Calcium estimations in general refer to total calcium and usually show figures which are subnormal or relatively low in proportion to those of the phosphate.

In regard to acidosis, Lathrop[18] estimated the carbon dioxide combining power of the plasma in his case and found it to be 30% by volume, which fell to 19% before death. In Schoenthal-Burpee's[19] case there was an average pH of 7·33. On clinical grounds, and on comparison with similar cases of adult nephritis, acidosis of marked degree may be assumed to be a very constant occurrence.

Renal Efficiency.

Urea concentration is always very markedly diminished, often not reaching 1%, while dyes are very poorly excreted, and similarly the chlorides (Alport[20]).

Morbid Anatomy.

The Bones.

Naked Eye Appearances. The characteristic appearances of bone are usually most marked in those bearing the body weight, or in the distal extremities of the radius and ulna. All bones are affected, including even the ribs and skull. The costo-chondral junctions in particular suffer marked disorganization. In severe cases the bones may be soft and elastic enough to bend slightly in the hand, and may be cut with a knife. They are also lighter in weight than the normal, and thinner in the middle of the shaft. Complete longitudinal section of the bone reveals the medullary cavity full of loose, pale and fatty marrow, and on the convex side approaches very closely to the surface, owing to the extreme thinness of the cortex. On the concave aspect there is usually some attempt at buttress formation, and the cortex is thicker than normal. Approaching the epiphysis, the cortex again becomes extremely thin, and a diminished zone of red marrow is apparent. The tissue adjacent to the epiphyseal cartilage is vascular and fibrous-looking, with white patches of fibrous tissue and dense calcification surrounding outlying islands or partial islands of cartilage which have not quite lost their continuity with that of the epiphysis.

The epiphyseal cartilage itself is much widened and has a very wavy, irregular proximal border, but a fairly straight distal edge, whilst laterally it may project beyond the curved contour of the shaft. The cauliflower-like projections of the epiphyseal cartilage into the substance of the metaphysis result in a heavy aggregation of vitreous-looking material, and are very characteristic of renal dwarfism. When such a change is very marked on one side of the metaphysis, there is frequently collapse of the weakened bone. Such a

change seems to be most frequent at the distal extremity of the radius or tibia. Though not always, this deformity is frequently associated with a straight, if thin, diaphysis. In the case of recent collapse, hæmorrhages are present, but they are subsequently organized during repair and add to the fibrous tissue element. The nutrient vessels share in the general hypertrophy of the arterial system, if such a change is present.

Histopathology. A section through the shaft shows the cortex to be thin and the periosteum thickened. There is an excess of fatty tissue in the medulla, and blood vessels have a thickened middle coat. In the spongy end of the shaft the trabeculæ as a whole are thin and fragile. Approaching the epiphyseal line and osteoid zone, they become more and more imperfect, with a few flattened "drone" osteoblasts applied to their sides. The impression given is one of chemical decalcification in which, unlike the decalcification of hyperparathyreoidism, osteoclasts play no part. More distally is an irregular mixture of outlying and deformed cartilage cells and osteoid tissue, intermingled with old and recently-formed fibro-cellular connective tissue, into which the irregular projections of true epiphyseal cartilage make their way. The proximal lines of these cells themselves are of various shapes and sizes, unlike the regular rows seen in nutritional rickets. No doubt the collapse, if present, is mainly responsible for this. A certain degree of round cell infiltration is often present in association with the connective tissue. The vault of the skull as a whole is considerably thickened, as in early Paget's disease, but there may be round or oval patches of rarefaction near the base, comparable to the craniotabes of ordinary rhachitis. On section, the cortex is seen to be thinned and the diplöe widened. The texture of the medulla is soft and may even be cut with a knife. Bossing is not common, nor any change in the sutures. Under the microscope the trabeculæ are thin and weak-looking.

The Kidneys.

The kidneys are usually more contracted and fibrotic than in the most extreme grade of chronic interstitial nephritis seen in the adult, even including those of the Rose Bradford type. The average total kidney weight varies between 12 and 100 grammes, according to the age of the child, and the size of the organs rarely rises above one-seventh of the normal. They are as a rule approximately equal in size, and are to be found in their normal positions. The surface is rough or coarsely granular and small cysts project from the surface. No cases of congenital hydronephrosis have been described, though the pelvis may appear relatively large (Case I). On section, the capsule is usually thick and adherent, so that it is stripped with difficulty, and small pieces of cortical tissue are torn off with it. The kidney substance is tough and leathery. The cortex is reduced to a very thin rind, so that the medullary rays appear almost to reach the surface. Tiny cysts may also be present near the capsule. The pelvic fat is increased to compensate for the reduction in functioning tissue. The blood vessels are usually prominent and gaping. Infection of the pelvis is sometimes present with a thickened granular or vascular epithelium.

From a careful histological comparison of the kidneys of Case I with those of two children dying of chronic nephritis without dwarfism, I have been unable to discover any feature of glomerulus, interstitial tissue or special tubule which appears more frequently in one type than in the other. There may be an exception in that in the kidneys of dwarfed patients it is the tubules as a whole which appear to bear the brunt of the damage. In the "lead nephritis" of Queensland stunting of growth occurred in six of nine cases reported by Nye,[21] amounting in three to true dwarfism (up to 25%) of expected height. These kidneys show a marked "nephrosclerosis" with an extraordinary absence of glomerular involvement.

Changes in Other Organs.

Changes in the heart are those of concentric hypertrophy, and all vessels show a fibro-muscular increase in the middle coat, most marked in the kidney. Cirrhosis of liver and spleen has been rarely noted. Though the question has been carefully examined by many observers, no constant alteration has been ascribed to the ductless glands.

Causation.

Ætiology of the Renal Insufficiency.

In the child the powers of compensatory hypertrophy to overcome an initial defect or damage are greater than in the adult, so that the illness, though often beginning very early in childhood, may not become fully apparent for at least several years. The added strain of early puberty seems often to be the point at which the kidneys can no longer "hold their own", and finally break down functionally.

Family History. A family history is noted nine times. In Zundel's[22] case a father and brother are noted as being similarly affected. Two of Barber's[1] patients were brother and sister. Hellendall[23] reported two sisters showing the same condition.

Syphilis. Though diligently sought for even in the earliest cases, syphilitic phenomena in the blood and elsewhere have been notably absent.

Previous Acute Nephritis. Only three patients in the whole series are noted as having had acute nephritis. Scarlatina is never mentioned.

Previous Chronic Inflammatory Conditions of the Renal Pelvis. In two cases the renal sclerosis was apparently secondary to the bilateral formation of large pelvic stones. Pyelitis was also notably absent in practically all patients.

Focal Infection. By far the commonest site of focal infection in the child is the naso-pharynx. The commonest sequel to this, as far as the kidney is concerned, is an embolic bacterial nephritis (Moncrieff and Wyllie[24]), which apparently clears

up spontaneously, leaving no lasting damage in its train. The frequency of post-nasal infection in children is not reflected in a frequency of renal damage.

Congenital Nephritis. The closer one examines the infancy of some of these patients, the more one becomes convinced that they suffered from some form of toxæmia in their infancy, capable of exerting a restraint on their mental and physical development. In a number great thirst was noticed almost from birth. Three possibilities have presented themselves as explanations for these infantile cases.

(*a*) Partial Agenesia of the Kidney. Lack of fusion between the portion of the blastema and the Wolffian duct outgrowth which go to form each tubule, would result in a large portion of the renal cortex being thrown out of action.

Maldevelopment of pronephros and mesonephros is relatively common, and there is no reason to suppose that these faults may not sometimes affect the more highly differentiated metanephros, with connective tissue occupying the place of the degenerated tubular remnants.

Figure X illustrates the cortex of a kidney of a child dying at the age of seven weeks, apparently from uræmia, in which the total kidney weight amounted to only 3·6 grammes. The photograph clearly illustrates the young fibrous tissue surrounding degenerated convoluted tubules and healthy-looking glomeruli, while even the smallest of the vessels shows marked muscular hypertrophy.

This may represent a precystic stage of congenital polycystic disease of the kidney or an extreme form of what may in later life have been the basis of renal dwarfism.

(*b*) Renal Abiotrophy. Abiotrophy has not been generally recognized to occur outside the central nervous system, though as far as the liver is concerned this explanation has been offered for both Wilson's, and some cases of Banti's, disease. Whether any cases of renal dwarfism can include such a pathology is very doubtful.

Intrauterine Nephritis. In regard to the possible late effect of the toxæmias of pregnancy on the child, it is becoming more generally admitted that preeclamptic toxæmia or eclampsia, even of moderate severity, may frequently leave in its train an "occult" or more rarely a frank chronic nephritis. Various observers state this proportion as being from 3% to 10%. I found that of fifty-seven *multiparæ* admitted to the Women's Hospital, Crown Street, Sydney, from 1919 to 1929, with toxæmia of marked degree, whose children had survived, two gave a history of a similar attack in a previous pregnancy. Of nineteen of these patients who accepted my invitation for estimation of their renal function, five had signs of renal insufficiency and four gave a history of a subsequent pregnancy with toxæmia.

Many infants of toxæmic mothers are born dead or live only a few hours after birth. Many others are obviously suffering from toxæmia with feeble respirations and pulse, are pallid or even œdematous. They may have convulsions, purpura or albuminuria. It is a reasonable assumption that the toxin, having once passed the placenta, would selectively affect the liver and kidneys of the fœtus, which are even more vulnerable and delicate than those of the mother. Though the child may apparently make a rapid recovery, it is possible that the reserve of the kidney has been permanently reduced and that the damage caused may be the starting point of an insidious and expansive vicious cycle, resulting ultimately in a frank chronic nephritis and its sequelæ. Such being the case, dwarfism and bone changes may be expected to appear after a considerable latent period. Of fifteen cases investigated from this standpoint, in six there was a history of maternal illness.

I examined twenty-six children whose mothers had been admitted to the Women's Hospital, Crown Street, Sydney, suffering from a moderate to severe grade of toxæmia. Some had been premature and weakly, some had exhibited the various signs noted above, others seemed quite unaffected. The children as a whole were particularly healthy and well, even above the average, notably those of the eclamptics. No evidences of any bone changes, dwarfism or renal insufficiency were found. There is, therefore, no one cause of juvenile chronic nephritis, and so of dwarfism of this type. The truth of these suggestions is very difficult to prove with the small amounts of clinical material available.

Ætiology of the Bone Changes.

Only part of the total calcium of the blood is available for bone formation, and amounts to five or six milligrammes per 100 cubic centimetres of blood plasma. Concentration of this ionized fraction is regulated by a system of buffers, including phosphate and carbonate ions which react to form insoluble and soluble intermediate calcium salts. Similarly, the portion of phosphate ionized and made available for bone deposition varies greatly with the pH of the plasma. In the limits of pH compatible with life only a very small fraction exists as the tertiary phosphate ion, and a change in pH from 7·2 to 7·4 almost halves this amount. According to both Howland's[25] and Robison's[26] theories, bone formation depends, among other factors, on the pH of the medium. Shipley, Howland and Kramer[27] showed that calcification in thin slices of normal bone *in vitro* occurs from within outwards, and only in solutions containing calcium and phosphorus in proportions and at a pH approaching those of normal serum. Robison has isolated an esterase from growing bone which has the effect of hydrolysing the hexose phosphates of the plasma and increasing the local phosphate concentration. It is suggested that possibly the osteoblasts do their work by forming a zone of increased alkalinity, which favours the process. As regards bone absorption, the physico-chemical actions described are all reversible, depending on the local carbonic acid concentration, the total number of tertiary phosphate ions and the pH of the medium.

Thus Ross and Scriver[28] have shown that administration of ammonium chloride, by producing an acidosis, can cause sufficient mobilization of "free" calcium from its reservoirs to relieve an attack of infantile tetany, but Kramer[29] points out that this treatment, if continued, will aggravate any condition of bony porosis, similar to that caused by repeated injections of an active parathyreoid extract (Greenwald and Gross[30]).

In chronic azotæmic nephritis there is a retention of acid phosphates. Theoretically, this should cause a corresponding diminution in the number of blood calcium ions if the postulate of Freudenberg and György[31] is to remain true. Thus:

$$\frac{(Ca^{++})\ (HCO_3^-)\ (HPO_4^-)}{H^+} = K$$

Boyd, Courtney and MacLachlan[32] have found constantly low calcium values in azotæmic nephritis, and de Wesselow[33] states that the higher the retention of phosphorus in nephritis, the lower is the calcium in the serum.

In renal dwarfism there is present a nephritis with a particularly well marked azotæmia, frequently a normal blood calcium and a raised plasma phosphorus and phosphatase, and a very definite acidosis. Or, in other words, if the blood calcium remains normal, as it often does in renal dwarfism, the increase in phosphate ions must be accurately balanced by the degree of acidosis. If such occurs in the child with the bone ends ununited and the nervous system in a relatively unstable condition of excitability, this means there are at least two mirrors which can reflect clearly any deprivation of active calcium, the images being (*a*) rickets, (*b*) tetany. In renal dwarfism the former is common, the latter rare. The only possibility by which this approximately normal level in the calcium of the blood can be maintained, is by continuous mobilization of calcium from the bones. There apparently exists, under the influence of the raised blood phosphate, a continual washing out of the calcium from the bones, and this is facilitated, as the observations of Ross and Scriver[28] have shown, by an acidosis which mobilizes calcium ions from the calcium reservoirs and favours the ionization of the remainder of the calcium not yet finally bound to protein. The biological intention of this process is almost certainly, as Parsons[13] suggests, to prevent for as long as possible the onset of tetany, which is so much more easily produced and of more serious consequence in the child than the adult. Bone formation is the least vital rôle which calcium has to play in the body economy, and recently deposited calcium phosphate is most readily made available before it is fully incorporated into living bone. The free calcium, like the sugar of the blood, is maintained at a constant minimum until the last, notwithstanding what depletion may occur in its stores.

The apparent normal or even raised figure shown in some of these cases cannot be taken as an indication of the true ionic value, since tetany can occur, though rarely. The calcium, therefore, in these cases must include only a relatively small amount of the free ionic form, or as Parsons puts it, "the serum calcium functions as low calcium".

Tetany in Renal Dwarfism.

The work of Loeb, Matthews,[34] and others has shown us that tetany is not so much dependent on the total calcium content of the plasma as on its "free" fraction, which is the physiologically active portion in preventing the appearance of tetany, as it is in the deposition of bone. They have shown that the excitability of motor nerves varies directly with the ratio $\frac{Na + K}{Ca + Mg}$. In ordinary rickets tetany can occur only from a diminution in the active calcium ions, as the remainder are unaffected, as has been demonstrated by Howland, Kramer and Tisdall.[35]

Tetany can be produced experimentally, among other ways, by the deprivation of the parathyreoid hormone, which causes a rise in acid-soluble phosphorus (Greenwald[36]), and by the injection of alkaline phosphates (Binger[37]). Klercker and Odin[38] produced the same effect in children by the administration of normal and alkaline phosphates, and where latent tetany was already present, severe spasms followed. Schoenthal and Burpee[19] have shown, however, that similar treatment of a patient with renal dwarfism had no such effect, the phosphate scarcely being absorbed at all (as occurs in ordinary rickets), a further provision of Nature against this exhausting complication. Binger found further that an injection of acid phosphates into dogs also lowered the blood calcium, but did not produce tetany. This emphasizes the difference between the utilization of calcium in the grown and growing organism. Tetany can be produced more readily in the latter, but the presence of an acidosis may prevent it by making more free calcium ions available. Thus Lathrop[18] found that in his case which had a carbon dioxide combining power in the plasma of 30%, although the total figure for calcium was very low (five milligrammes per 100 cubic centimetres), yet the "free" calcium was at 3·8 milligrammes per 100 cubic centimetres, above the tetany level.

Flux between healing and relapse also occurs in the bones in renal dwarfism, and Parsons and Duken[39] are the only observers who have traced the accompanying changes in the blood. Coincident with an unexplained rise in blood urea, the blood phosphate rises with its concomitant mal-effect on the blood calcium. As the child recovers, the phosphorus is better excreted, the blood calcium level rises and the bone changes begin to improve. As the child grows older (about sixteen years), growth is slower and so less calcium is required. Tetany is less frequent and bone changes less extreme. Complete healing with healthy-looking adult bone

can occur, as Barber has shown in a girl aged twenty, and Parsons in a boy aged sixteen.

Are the Bone Changes Those of True Rickets?

Clinician, radiologist and histologist have all been deceived into identifying renal dwarfism with ordinary rickets, and it is interesting and useful to consider at this point whether it conforms to the biochemical criteria of the latter. The product obtained by multiplying the total phosphorus and calcium values in milligrammes per hundred cubic centimetres (Howland's constant) has frequently been cited as a criterion of the plasma for the presence of active rickets. Normally having a value of 50, active rickets is supposed to be present when it falls to 30 or below. This rule fails in the case of renal dwarfism, but when the product is made that of the ions only and expressed as the negative logarithm of molecular concentration, as is done with the hydrogen ions in expressing the reaction of a solution, the figure becomes 24·1. This is called the "*p*" product, above which active rickets is usually present. This method, described in a paper by Holt, La Mer and Chown,[40] is, of course, much nearer the truth, but yet when Parsons estimated the product in each of his cases they were invariably less than 24·1, that is, rickets should have been healing or absent. This figure, however, has been worked out on the assumption that the pH of the blood is 7·35. I have already mentioned that Holt showed that even the smallest variations in pH affect considerably that proportion of the blood phosphate ions which are so important in bone formation. Parsons, using the method of Druker and Cullen,[41] determined the pH of one of his cases before determining the "*p*" product. Even then, the result was just below 24·1. By using this method, however, he was able to refute Lathrop's statement that the bone changes are not rhachitic, by proving this author's own case to have a "*p*" product quite within the zone for active rickets.

Quite apart from radiological and pathological evidence, it is thus certain that the changes of renal dwarfism are those of true rickets, and indistinguishable, as regards the florid type at any rate, from ordinary florid rickets of Wimberger. It thus appears that Shipley[27] was right in saying that "a true renal rickets exists, and rickets under certain conditions may have an endogenous origin".

Other Theories of Causation.

A Possible Endocrine Factor.

It is known that at least two ductless glands have specific effects on calcium utilization and bone growth, namely, the parathyreoids and the hypophysis, but naked eye and microscopical examination of the former and radiological visualization of the latter have failed to reveal any hypertrophy or other abnormality. No change has been noted in the ovaries.

Ashcroft,[2] in the paper referred to, says that "sufficient evidence has been found to warrant the suggestion that the disease may be due to a fibrosis of the suprarenal gland". Certainly the photomicrograph of the adrenal cortex in his case suggests considerable atrophy, which may have been due to a narrowing or atheroma of the suprarenal arteries. Other observers, however, have failed to find any such change, and Swingle[42] has shown that though total removal of the adrenals causes a sudden elevation of blood urea and phosphorus, the smallest amount of cortex remaining suffices to prevent it. The facies, dry skin *et cetera* have also been described as cretinoid in character, but no observer has noted any gross change in the thyreoid. One estimation of basal metabolism has been done in America, with a normal finding.

A Possible Nutritional Factor.

It is a new fact of some interest that in practically all reported cases the patients are children of the public hospital class in large industrial cities. Cases I, II and III were all from homes with poor hygienic surroundings. It is impossible, therefore, in some of the youngest patients, to exclude a preceding or coincident nutritional rickets. It may be possible that some of these infantile patients develop an ordinary rickets in the period of election, six to eighteen months, during which time diet and habits are not placing any strain on the renal reserve, which also at this time may not be wholly exhausted by a developing lesion. The lesion established, full healing is prevented and the disease made worse by the now permanently altered metabolism consequent on the full development of the renal sclerosis, which probably interferes with the usual mode of action of our anti-rhachitic measures. Such an explanation would be perhaps why these cases are rare in Australia, where florid rickets is uncommon, and so frequent in the hospital class of large industrial cities of Europe. The contrary evidence is very strong, and in the Schoenthal-Burpee case cod-liver oil was given from three months and the diet was always well balanced. In the older cases also renal rickets develops and persists in the presence of an adequate supply of calcium, phosphorus and vitamin *D* in the dietary, and under satisfactory hygienic conditions. If further proof is needed against this factor in older children, it is that Parsons has shown a patient to become worse under prolonged treatment by ultra-violet light, apparently due to an increase in phosphorus absorption.

The Radiology of Renal Dwarfism.

Parsons produced conclusive evidence that his cases could be divided into at least two groups, which he called the "florid" type and the "woolly" type, distinguishable by observation of the wrist epiphyses alone.

The Florid Type. In the florid type the metaphysis is concave or even flask-shaped, widened, and with considerable recession from its appropriate epiphysis. This is the well known picture of nutritional rickets in an active phase, "the florid rickets of Wimberger", and the two are indistinguishable by this means. There is no clinical counterpart to

ILLUSTRATIONS TO THE ARTICLE BY DR. KEMPSON MADDOX.

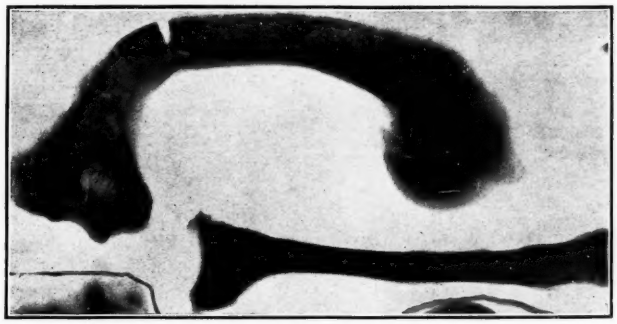

FIGURE III.
Post mortem radiograms of bones from Case I. Note the extreme porosity and thinness of shaft resulting in a characteristic stippled appearance, absence of cortex, sharp definition to distal edge of epiphyseal line and irregular, vague proximal border. The apparent fracture is an artifact.

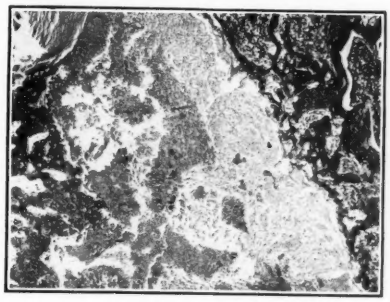

FIGURE IV.
Appearances at the lower end of the femur in Case I (×3). Note the cartilaginous extension into the shaft at the upper left-hand corner, the poorly formed trabeculæ and degenerate cartilage.

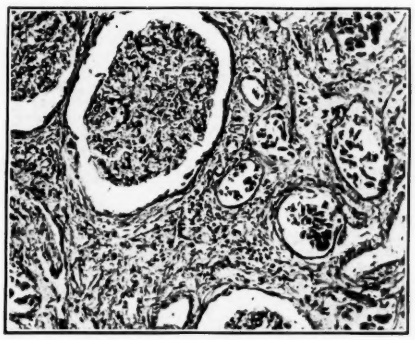

FIGURE V.
Hypertrophied glomeruli in renal cortex of Case I. Note hyalinization of tuft and adhesions to capsule, also collections of small round cells in the upper left-hand corner, with fibrosis of the adjacent blood vessels (× 95).

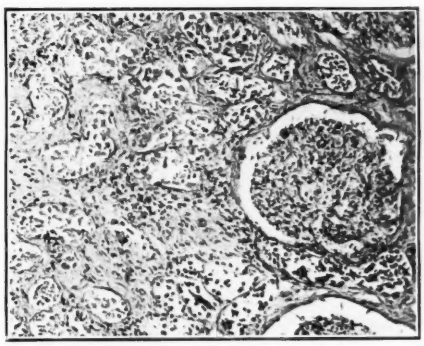

FIGURE Va.
Microscopical appearances of renal cortex in Case I, showing dense scar tissue containing compressed degenerate tubules (×96).

FIGURE IX.
Radiogram of skull in Case III. Note the woolly appearance in the cortex of the vault due to a fine degree of stippling. Note also "the baker's fingerprint" appearance towards the occiput.

ILLUSTRATIONS TO THE ARTICLE BY DR. KEMPSON MADDOX.

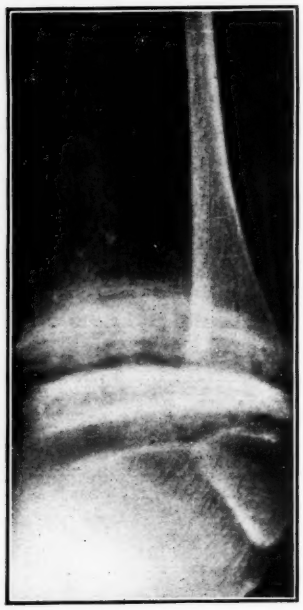

FIGURE VIII.
Radiogram of Case III, showing appearances at the lower end of the tibia which are typical of a moderate case of renal dwarfism. The serration is very well shown, surrounded by dense sclerosis marking previous attempts at healing. Note also the generalized rarefaction of the shaft.

FIGURE XIII.
Radiogram of P. 7 one month after nephrectomy. Note apparent increase in normal lipping and uniform dense haze at metaphyses.

FIGURE XV.
Autopsy radiogram of P. 30, showing disappearance of abnormal appearances at the bone ends and healthy texture of the shafts, which, however, remain curved.

FIGURE X.
Microscopical appearances in a case of congenital nephritis, with death at seven weeks. Note the permeation with young connective tissue, fairly healthy glomeruli, degenerate and dilated tubules, collections of small round cells, and muscular hypertrophy of vessels.

FIGURE XIV.
Autopsy radiogram of P. 31. Note the thin quality of the shafts and commencing clouding of metaphyses.

FIGURE XVI.
Autopsy radiogram of P. 9, suggesting apparent complete disorganization of lower epiphysis of tibia not confirmed microscopically.

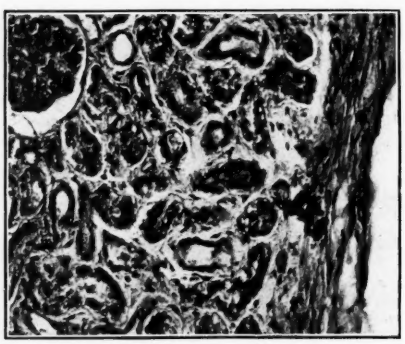

FIGURE XVII.
Renal cortex of P. 12 (×160). Note shrivelled character of superficial zone invaded by connective tissue from the thickened capsule. Many tubules have lost their epithelium or this is represented by odd nuclei. Two glomeruli are shown, one (on the left) relatively undamaged.

this appearance, except that it tends to occur slightly more frequently in the younger children, and in a pure form is a less common finding than the other type. Osteoporosis occurs, and bending with buttress formation is no more usual with this group.

The Woolly Type. The woolly type shows the changes which are now considered so characteristic of renal rickets alone. The typical changes occur in the region of the proximal border of the epiphyseal cartilage. Instead of being straight or concave, this edge is irregularly serrated and has a ragged, rat-eaten appearance extending right across the widened metaphysis. In some cases rounded gaps of considerable size appear and connect with similar areas of rarefaction deeper in the shaft. The metaphysis extends over a much wider area than in ordinary rickets, and an apt description of the metaphysis is that "it is like a wooden post rotted by the earth". The radiograms of Case III (Figure VIII) show the typical changes just described. A further stage of this process is collapse of this irregular cartilage, giving the appearance of an impacted fracture. The rarefied areas in the shaft probably represent the remains of old epiphyseal lines and correspond to the cartilaginous islets. The shaft as a rule is not quite so atrophic as in the "florid" type, and is consequently straighter. There is in the blood apparently no biochemical equivalent to these types.

The changes are frequently asymmetrical and do not occur to the same extent in all bones, although conforming to one type in the same patient. They are usually best seen at the wrist or ankle. The changes at the costo-chondral junctions are never distinguishable from those of ordinary rickets. Signs of old fractures, usually about the centre of the shaft, are sometimes noted, but healing (of sorts) has usually occurred.

Healing occurs at the bone end. The whole texture of the metaphysis improves in definition, and little remains to show what occurred, except a fine transverse line at some distance above the line of epiphyseal union, which marks the site of the old trouble. Between these lines the trabeculæ appear finer and smaller. Sometimes these lines are prominent and numerous, as in Figure VIII of Case III, and undoubtedly mark previous attempts at healing, or perhaps relapses in the disease, as toxic and nutritional factors can cause similar appearances (Harris).

Other inconstant changes noted by various observers are an extreme porosity of the finger phalanges (Karshner[43]), fusion of the vertebræ (Smith and Walsh[44]) or carpal bones (Ashcroft), delay in ossification of metacarpals (Schoenthal and Burpee). Changes in the skull are characteristic and deserve special comment. The vault is most affected and shows gross thickening to twice that of normal. It has a typical fuzzy appearance, and all distinction between cortex and medulla is lost (Figure IX). Radiographers, confronted with these appearances for the first time, repeatedly describe them as those of *osteitis deformans.* Areas of irregular rarefaction and condensation may exist near the base, and there is usually a generalized "stippling", which may reach an extreme degree. No abnormality has yet been discovered in the pituitary fossa.

Differential Diagnosis.

Early diagnosis is not easy, owing to the insidious onset, but once an observer has seen a fully developed example of the disease it is unlikely that he will fail to recognize it again. It causes usually an extreme degree of dwarfism, which in itself should arouse suspicion. It must be distinguished from two separate groups of diseases: first, the remaining causes of dwarfism and infantilism; secondly, other conditions resulting in similar bone deformities.

Diabetes Insipidus. Experience shows that several cognizant clinicians, such as Zundel, have at various times confused renal dwarfism with *diabetes insipidus.* It can be readily understood how early writers, such as Carter,[45] confused these two conditions. It may be of help to note here the essential difference between them: In *diabetes insipidus,* (i) dwarfism is not marked; (ii) the urine is free from albumin, and there is no elevation of the blood urea; (iii) there is no alteration in the bones, either clinically or radiographically; (iv) many cases give a positive Wassermann reaction in the blood; (v) the excessive urinary output is immediately lessened by injection of an active extract of the posterior lobe of the pituitary gland, for example, pitressin; (vi) there is no cardiovascular hypertrophy or retinitis.

Other Causes of Dwarfism. Renal dwarfism has been confused with dwarfism due to other causes, such as cretinism, achondroplasia, congenital heart disease, pancreatic disease, cœliac disease, *osteogenesis imperfecta,* progeria, ateliosis, and the Lorain and Brissaud types of dyspituitarism. These are all readily distinguishable, however, by their characteristic features, but tests for renal efficiency and urine tests are at once conclusive, combined, if any further doubt exists, with radiograms and an estimation of the blood phosphorus.

Juvenile Diabetes Mellitus. The dry skin and tongue, constipation, retinitis, thirst and polyuria are all features common to both renal dwarfism and congenital *diabetes mellitus.* Even glycosuria has been observed in renal dwarfism, but a sugar tolerance test would at once decide the question.

Hereditary Ectodermal Dysplasia. In hereditary ectodermal dysplasia there is a want of appearance of some or more ectodermal elements, so that the children who are frequently dwarfed, have sparse hair, a few pointed teeth and a very dry skin; as a result, no sweating occurs and hyperpyrexia may

TABLE SHOWING DETAILS OF EXPERIMENTS.

Litter No.	Dog No. "P."	Approximate Age at First Oper'n.	Sex.	Weight at First Operation in kilograms.	Weight of Excised Kidney in grammes.	Duration of Irradiation Minutes of Standard dose	Period between Operations (days).	Period between Irradiation and Death.	Growth.	Blood Urea. Milligrammes *per centum*.	Complications and Cause of Death.	Radiogram.	Blood Hæmoglobin. Percentage.	Weight of Irradiated Kidney at Death. (grammes).	Histological Damage to Kidney.	Remarks.
I	1	5 mths.	F.	3·74	24·0	5	14	—	Unaffected.	42–75	——	Shows rickets before nephrectomy.	50	—	——	Living. Bones fully healed.
I	2	5 mths.	F.	3·8	25·2	5	18	7 mths.	Unaffected.	48–106	Infection of back. Compound fracture of leg.	Shows rickets.	40	43·6	Slight.	——
—	3	6 mths.	M.	5·4	29·2	5	27	28 days	—	—	? fatty degeneration of liver.	—	—	33·0	Grave.	Liver extremely fatty and mottled.
II	4	2 mths.	F.	1·6	9·7	5	—	10 days	—	—	Tetany-like fits.	—	—	9·7	Acute disorganization.	——
—	5	5 mths.	F.	4·0	18·9	5	—	10 days	—	—	? Acute renal failure.	—	—	18·6	Coagulative necrosis of cortex.	Died at night, suddenly.
—	6	3 mths.	F.	3·5	20·1	10	31	35 days	—	34 *post mortem* 549	? Acute renal failure.	Shows rickets before operation.	—	14·5	Marked, associated with much fatty degeneration.	Death like latent uræmia.
III	7	7 wks.	M.	1·3	13·3	5	17	6 mths.	Affected.	30–96	Laryngeal hæmorrhage.	Shows rickets before operation.	—	26·6	Signs of old damage, *plus* regeneration.	*Vide* text.
III	8	7 wks.	M.	1·0	7·4	5	—	5–10 hrs.	—	—	Pneumonia.	—	—	7·6	Acute inflammatory reaction and necrosis.	Probable earliest change.
III	9	7 wks.	M.	1·1	8·5	5	14	27 days	Affected.	132–144	Cancrum oris.	Gross disorganization.	32	9·7	Generalized damage and toxic change.	Radiographic signs not confirmed by histology.
III	10	7 wks.	F.	1·2	8·8	5	14	26 days	Affected.	*Post mortem* 233	Peritonitis.	Shows rickets before operation.	—	8·3	Great cortical damage. No interstitial increase.	Esbach, 4 grammes.
IV	11	11 wks.	F.	2·8	16·8	10	—	12 days	—	—	? Cardiac failure.	—	—	17·4	Extensive tubular destruction. Glomerule fairly normal	Died with anasarca.
IV	12	11 wks.	F.	2·5	15·7	5	14	24 days	Affected	75 *Post mortem* 141	Cancrum oris.	Shows rickets.	—	18·7	Patchy necrosis. Majority glomeruli destroyed.	Esbach, 2 grammes. Cholesterol, 161 milligrammes %. Urine urea, 6%.
IV	13	11 wks.	F.	3·0	18·7	5	14	32 days	Affected.	30 *Post mortem* 90	Fits.	Shows rickets.	—	20·6	All elements badly involved.	——
V	14	11 wks.	F.	3·6	20·7	5	20	21 days	—	50	Liver abscess. Broncho-pneumonia.	Shows rickets *post mortem*.	—	22·7	Extreme damage to all parenchyma.	Apparently death from toxæmia.
V	15	11 wks.	F.	3·1	13·7	5	14	14 days	—	—	Anæsthetic death.	—	—	16·8	Œdema and acute necrosis.	——
VI	16	5½ wks.	F.	1·1	7·3	5	10	20 days	Affected.	*Post mortem* 333	? Acute renal failure.	—	—	5·45	Extensive involvement of all elements.	No radiogram taken.
VI	17	5½ wks.	F.	1·0	9·1	5	10	10 days	—	—	Anæsthetic death.	—	—	8·1	Œdema. Amorphous areas.	No radiogram taken.
VI	18	5½ wks.	F.	1·0	9·0	5	10	18 days	Slightly affected.	232	Fits. ? Acute renal failure.	—	—	8·15	Widespread atrophy tubular epithelium. Young fibrous tissue visible. No normal glomeruli.	Direct Van den Bergh reaction positive. Andrewes's reaction not obtained.
VII	19	9 wks.	M.	1·0	7·05	6	—	16 days	—	—	Cachexia.	—	—	7·1	Generalized cortical damage of moderate degree.	Poorly nourished from start.
VII	20	9 wks.	F.	1·25	8·1	6	—	11 days	—	—	Cachexia.	—	—	7·85	Generalized cortical damage of moderate degree.	Poorly nourished from start.
VII	21	9 wks.	M.	1·5	12·0	6	—	15 days	—	—	Cachexia.	—	—	10·9	More severe glomerular damage.	May have had renal failure.
—	22	10 wks.	F.	1·5	7·25	6	—	10 min.	—	—	Operative shock.	—	—	8·9	Slight beyond œdema and hæmorrhage.	——
—	23	9 wks.	F.	2·1	30·6	10	9	22 days	Not affected.	*Post mortem* 487 Cholesterol 250	Fits and coma with œdema. Certain renal failure.	Clear.	—	5·85	Fibrosis, early and great destruction of all elements.	Cardiac puncture, 50 cubic centimetres; partial recovery, 120 cubic centimetres; death. Andrewes's reaction obtained.
—	24	10 wks.	F.	2·2	10·8	10	9	20 days	Affected.	—	Intussusception.	Clear.	—	9·5	Nearly all tubular epithelium gone. Infiltration commencing.	No examination of blood made.
VIII	25	8 wks.	F.	1·5	—	6	—	12 days	—	—	Worm-infested.	—	—	—	No autopsy.	——

TABLE SHOWING DETAILS OF EXPERIMENTS.—*Continued.*

TABLE SHOWING DETAILS OF EXPERIMENTS.—*Continued.*

Litter No.	Dog No. "P."	Approximate Age at First Oper'n.	Sex.	Weight at First Operation in kilograms.	Weight of Excised Kidney in grammes.	Duration of Irradiation Minutes of Standard dose	Period between Operations (days).	Period between Irradiation and Death.	Growth.	Blood Urea. Milligrammes *per centum.*	Complications and Cause of Death.	Radiogram.	Blood Hæmoglobin. Percentage.	Weight of Irradiated Kidney at Death. (grammes).	Histological Damage to Kidney.	Remarks.
VIII	26	8 wks.	M.	1·7	7·2	6	—	10 mins.	—	—	Operative shock.	—	—	9·25	Œdema and cloudy swelling of convoluted tubules.	—
—	27	10 wks.	F.	1·1	8·25	6	—	8 days	—	—	Cachexia.	—	—	7·9	Widespread damage throughout cortex.	—
IX	28	7 wks.	M.	2·2	13·1	5	17	4½ mths.	Unaffected.	36–37	? Distemper.	Slightly shows rickets.	—	30·2	Half tubules gone. Glomeruli severely affected, except close to medulla.	Probable early compensation. Rayed twice through skin.
IX	29	7 wks.	M.	2·2	14·4	5	17	6 wks.	Affected.	102–140 *Post mortem* 87	Anæmia.	Atrophy and curvature Epiphysis slightly shows rickets. Shows rickets.	20	17·5	Similar to P. 28.	Bled from pancreas. Hypotonia.
IX	30	7 wks.	M.	1·7	13·7	5	20 days	4 mths.	Not affected.	24–60 Cholesterol 144	Pneumonia.		—	28·2	Capsule thick. Evidences of regeneration and fibrosis. Glomeruli nearly all damaged.	*Vide* chart.
IX	31	7 wks.	M.	1·7	11·4	5	7	4½ mths.	Not affected.	74–131 *Post mortem* 300	Acute renal failure. Terminal pneumonia.	(1) Slightly shows rickets. (2) Definitely shows rickets *post mortem.*	—	26·5	Much œdema. Nearly all glomeruli inactive? Tubules hypertrophied.	Rayed through skin a week before death. Esbach, 4 grammes.

follow. There are no urinary or bony changes, and adult life is often attained.

Addison's Disease. The pigmentation, pallor, dry skin and muscular weakness of renal dwarfism may lead to a diagnosis of Addison's disease, but asthenia is not present to the same degree, blood pressure is not so low, nor does it respond so well to injections of adrenalin. Pigmentation of the mucous membranes has not been described in renal dwarfism.

Radiological Diagnosis.

In the examination of radiograms special difficulties may appear and the appearances be confused with those of: (i) ordinary rickets of the florid type, (ii) syphilitic diaphysitis, (iii) chronic osteomyelitis, (iv) scurvy, (v) other types of rickets, such as cœliac rickets.

It is well to remember that albuminuria may not be present at every examination, and must be sought repeatedly. If any doubt should arise, renal efficiency tests, such as the urea concentration, phenolsulphonephthalein and thiosulphate tests, should be employed without delay, and particularly if any operative interference is contemplated. All cases of "late" rickets should be suspected of renal defects until proved otherwise, and it is only in this way that disaster will be avoided.

Course and Prognosis.

The course of the disease is determined by the rate of advancement of the underlying nephritis and the degree to which compensation can occur in young persons. Apart from this latter factor, the prognostic guides are the same, but of less value than those in adult nephritis. The place of bone changes in this sphere is of little value, except as a rough guide to the phosphorus retention. Barber has said that death usually occurs within two to three years of the onset of deformity. He records, however, one case which remained ten years in an active state before death intervened at the age of twenty-three, and refers to another patient who had had the disease for seventeen years but still had no cardiovascular hypertrophy, a slight *genu valgum,* a blood urea of 113 milligrammes per 100 cubic centimetres, and urea concentration of 0·7%. There are many other exceptions to this rule, and Parsons describes a patient, well at twenty years of age, both clinically and radiographically, who had quite severe rickets at sixteen. When deformities occur in infancy, the prognosis is most serious of all. The strain of puberty often shows itself by a rapid increase in bone changes, and death occurs with greatest frequency in the second decade. The average age of death in fourteen unselected cases was sixteen years. In all long standing cases sudden increases, of obscure ætiology, in blood urea (up to 226 milligrammes per 100 cubic centimetres in one of Parsons's cases) and phosphates occur from time to time, but partial recovery may follow just as quickly. Fits also do not seem to have the same prognostic significance as in the

adult. Similarly, poor results of the urea concentration test do not necessarily mean an early demise. Very large blood urea readings (up to 600 milligrammes per 100 cubic centimetres) are found just before death. Non-protein nitrogen estimations are more reliable than those of blood urea. When a marked elevation in urea or non-protein nitrogen persists (100 milligrammes per 100 cubic centimetres or more), with a rising blood pressure and progressive retinitis, death may be expected within a few months.

Complications.

Uræmia. Uræmia is so common that it may almost be considered a part of the disease, and has already been discussed.

Tetany. The causation and significance of tetany have already been mentioned. It was terminal in Case I, and probably in many other patients who died out of hospital, but in a latent form is probably present during phases of increased azotæmia, if searched for. It was observed by Watson-Wemyss[46] during non-terminal epileptiform seizures, and during periods of increased headache and vomiting. Duken's patient, admitted to the hospital with tetany, recovered with healed bones, the phosphorus slowly falling to normal and the calcium showing higher values. György[47] showed that in his case a calcium-phosphorus ratio existed which was below the tetany level and yet no tetany followed. There are five cases in the literature which have shown frank tetany. Hunter (Goulstonian Lectures, 1930) states that he has observed opacities in the lens by the slit-lamp, as occurs in other examples of calcium deprivation.

Chest Complications. Bronchitis and pneumonia are noted in several histories and autopsies. The soft chest walls probably predispose to inefficient pulmonary ventilation, and nephritis, in the adult at least, is known to facilitate inflammatory invasion of this type.

Cerebral Hæmorrhage. Cerebral hæmorrhage is noted in two cases only. The frequent absence of cardiovascular hypertrophy and better resistance to degeneration in the child are sufficient explanations of its rarity.

The Skin. The skin is apparently liable to pyogenic invasion, and petechiæ may appear, as in the adult.

Treatment.

Unfortunately, little remains to be said in regard to treatment, the general treatment being the same as for any nephritis. It may be possible by a low nitrogenous diet, abundant fluid and free elimination, to lower somewhat a highly elevated non-protein nitrogen, and by protection from exposure and infections to ward off chest complications and uræmia. Organic drugs are of no use. Cameron[49] has shown that full doses of calcium, given by the mouth or intramuscularly, can temporarily raise to a slight degree the total calcium of the blood, and, combined with parathormone, may be used here to relieve a distressing attack of tetany. Calcium preparations, given by mouth, have been tried for long periods in renal dwarfism, but apparently have no power to arrest the course of the disease. This is only to be expected when one considers that the total calcium of the blood frequently approaches normal even in an actively progressing case, and there is usually an abundant supply in the food. Alkalis in amounts sufficient to have any effect on the acidosis must be large, and have only a temporary effect. Lathrop, however, raised the carbon dioxide combining power of plasma in his case from 16% to 24%, by volume, with alkalis given by mouth. Schoenthal and Burpee have shown that replacement of the depleted base and calcium is a logical method of treatment and can be achieved by an alkaline ash diet, together with calcium acetate. The phosphate contained in this mixture will do no good, but very little harm, as it is so poorly absorbed. They agree, however, that these measures must be employed continuously. During periods of infection or increased azotæmia with anorexia and diminished food intake there is a loss of base which can be prevented by parenteral administration of glucose, Ringer's solution and bicarbonate. In Schoenthal and Burpee's cases this treatment was followed by distinct clinical improvement, which was maintained to a certain extent by periodic blood transfusion. Parathyreoid, given by mouth, thyreoid and iodine have all been tried without effect. From Greenwald's work continued administration of an active parathyreoid extract may be expected to increase the bone changes.

Surgical treatment is of no use, as, apart from the danger of post-operative uræmia, the deformities frequently recur in a worse form. Where the patient has survived the operation, the legs have remained straight for a time, but soon give way, producing a grosser deformity than ever, so that these patients never walk again after the operation. Any interference in the mild cases should be done under local or spinal anæsthesia. Rest in bed during the acute stage or periods of relapse is necessary, and later, light supporting or correcting irons may help until the growth period is over.

An Experimental Attempt to Reproduce the Disease.

There has apparently been no experimental work done hitherto with particular relation to renal dwarfism, nor any attempt made to reproduce the disease in animals. Granting that chronic renal insufficiency is a necessary antecedent factor in the production of this condition, the problem resolves itself into the attainment of a suitable degree of renal failure in an animal sufficiently young as to be comparable with the human series.

Production of Nephritis by Exposure of the Kidney to X Rays.

Using rays of constant penetration on the dog, Hartmann, Bolliger and Doub[48] show that the kidney is relatively sensitive to powerful doses of X rays, and that repeated irradiation of the kidney

through the skin with hard X rays is capable of inducing fairly constant renal damage with clinical and functional results, including cardiovascular hypertrophy, similar, though not identical, to those caused by chronic nephritis in man.

Using a tube designed for therapeutic use in man, Bolliger, Laidley and Earlam[49][50] have administered a single, constant, direct, heavy dose of X rays to the exposed kidney in a large series of full-grown dogs. Animals receiving the same dosage of X rays, preceded or followed by heterolateral nephrectomy, pursued an almost unvarying course whereby death could be foretold with considerable accuracy.

In general, the course of the disease could be divided into three stages: (*a*) A stage of acute nephritis. The dog is little affected, but microscopical examination of the kidneys shows a marked exudative reaction involving all components. (*b*) A latent period and a period of tubular degeneration. After about a week of latency the kidney shows progressive degeneration and desquamation of the superficial tubules, which progress uniformly throughout the subcapsular zone until few nuclei remain. Meanwhile, the animal wastes somewhat, but otherwise appears quite well, while the levels of the blood urea, creatinin, cholesterol and carbon dioxide combining power of the plasma remain within normal limits. (*c*) A period of fibrous tissue invasion. In the smallest animals this period commences from the twenty-first to the twenty-second day after irradiation and marks the exhaustion of the renal reserve. From this time wasting becomes more rapid, sometimes disguised by slight œdema, thirst is increased, followed by dehydration, pallor, anorexia, dyspnœa, coma and death by the forty-ninth day after treatment. Histologically, the convoluted tubules are completely destroyed and are beginning to be replaced by an ingress of fibrous tissue from the thickened capsule, while the glomeruli remain apparently normal. The levels of blood urea, creatinin, cholesterol ascend rapidly to the death point. The carbon dioxide combining power of the plasma indicates an extreme acidosis. It appears that the size and weight of the dog are most important, in that the larger dogs in some cases reach a functional equilibrium. The regeneration is not observed until the twentieth to sixtieth day period. Constitution, sex, superficial wound infection and season do not affect the uniformity of the lesion. No cardiovascular hypertrophy, bone change or muscular hypotonia is observed. Pathologically, the lesion, whilst a distinct entity, most closely resembles that form of nephrosis in which an interstitial element ultimately makes its appearance. The interstitial tissue is mainly that of repair or replacement, and does not form the dense scars so common in the human disease. How far the cardiovascular changes recorded by Hartmann, Bolliger and Doub add to the production of fibrous tissue is difficult to say, but the hyalinization of glomeruli noted by these workers may be due to this factor.

Application of the Foregoing Results to the Problem of Experimental Renal Dwarfism.

Following on the above work it was felt that an inquiry into the effect of a nephritis which is capable of such accurate control, on the growth and general development of a young subject, that is, a puppy, would be of considerable interest, particularly as the identical X ray machine used above was available for the experiments.

Technique of the Present Experiments.

The technique of the experiments may be described shortly as follows:

General. It has been the endeavour throughout to secure whole litters at once and treat them simultaneously, in order to have some guide for comparison. Several litters were born in the laboratory and were nursed by their mothers for as long as possible (about five weeks). After weaning, they were fed with the remainder on a generous diet consisting of fresh, cooked and minced meat, biscuits, cow's milk, scraps and water. They were allowed plenty of exercise in the sunshine, and the majority received adequate daily amounts of cod liver oil.

Operative. The puppy has been selected for treatment at the earliest age at which it is capable of standing the operative interference (usually about six to ten weeks). The animal was then shaved and the skin cleansed, the anæsthesia was secured with tri-brom-ethyl alcohol ("Avertin") supplemented usually with a little ether by inhalation, as described in a previous communication.[51]

An incision was made in the loin, the kidney delivered externally, and its fatty capsule cleared. After screening the adjacent tissues and skin by means of a split lead plate, the kidney was exposed to the direct rays to be described, for a period varying from five to ten minutes. Afterwards the kidney was replaced and the wound sutured in layers with chromicized catgut. It was found to be impossible to perform the whole operation extraperitoneally, owing to the extreme thinness of this membrane in puppies. The procedure was practically bloodless. The animal was immediately returned to an isolated warmed kennel. About ten days later, when the wound had healed, nephrectomy was performed by the same route on the other side and the excised kidney weighed.

Quality and Quantity of the X Rays. The tube used was the identical one employed by Bolliger, Laidley and Earlam for their work with full-grown dogs, and was calibrated at that time by Dr. J. G. Stephens, who reported the quantity of radiation as measured with a Hammer dosimeter as 2·1 international *r* units per second at the surface of the preparation. The quantity of radiation was that emitted from the target of a Philips "Metalix" therapy tube, through which a current of 3·5 milliampères was passed at a potential of 130 kilovolts. A Seemann spectrogram revealed the shortest wave length as 0·11 Ångström unit, as confirmed by the Moll microphotometer, so that the peak voltage used was actually 112 kilovolts. All filters were removed and the target placed at a distance of ten inches from the surface of the preparation. In twenty-one cases (two-thirds of the series) the kidney was exposed to these rays for a period of five minutes, in seven cases for a period of six minutes, and in four cases for a period of ten minutes. In those cases in which additional irradiations through the skin were administered subsequently, the same tube was employed, but with the addition of a two millimetre aluminium filter.

Study of Clinical Appearances and Bone Changes. Observations began immediately after the second operation, and notice was taken of the general appearance, growth, weight, condition and appetite of the animal, together with the state of the muscle tone and posture, supplemented with photographs. Periodic measurements were taken of the total length of the animal from claw to claw, when fully stretched on a flat surface, together with estimations of the length of the radius and the circumference of the distal radial epiphysis. Radiograms

of the limbs were taken during the second anæsthesia, then periodically and after death.

Biochemical Observations. Biochemical investigations included frequent estimations of the blood urea level following the method of Marshall as described by McLean;[62] inorganic phosphate of the blood by the method of Benedict and Theis;[63] total blood calcium by the Kramer-Tisdall method as modified by Collip;[64] blood cholesterol after the method of Myers and Wardell.[65] Inquiry into the last named factors could not be performed so frequently as was desired, owing to the difficulty and danger of removing sufficient blood for all these purposes from a small puppy.

Histological. An autopsy was invariably performed to determine the cause of death, and slides were prepared in every case from the rayed kidney, irrespective of the time which had elapsed since treatment. In several instances the growing ends of the long bones, in particular the radius and tibia, were also preserved, decalcified, and the sections cut.

Results of Experiments.

General.

Thirty-one puppies were subjected in whole or part to the above procedures during the period April, 1929, to April, 1930. Of these puppies, one was alive at the time of writing. Of the remainder, three died within twenty-four hours of the first operation and nine were dead before the second operation could be performed. Seven died within ten days of the second operation, four died between this period and within three months of the second operation. Many more died before any interference could be made. The chief causes of death were operative shock, *cancrum oris,* hæmorrhage following vocal cord resection, and post-operative cachexia, due in two cases to sepsis. Two had tetany-like fits, apparently non-renal in origin, and four apparently died from an acute renal insufficiency. The ultimate mortality was thus very high, as it is particularly difficult to rear puppies under such circumstances. In spite of such misfortune, however, and owing to the rapidity of growth in puppies of this age, many survived sufficiently long for any effect the interference may have had on bone growth to be observed, and all provided useful material for histological purposes. Thus there were fourteen animals which survived both operations and lived for a sufficient period afterwards for any effect on bone growth and bodily development to become apparent.

Clinical Findings.

There was usually a fairly rapid recovery after nephrectomy, so that within a week or ten days the wounds were almost healed, and the puppies, still taking the same diet as before, were active and eating well. Superficial infection of the wound was common, but did not seem to affect the health or progress of the animal. Following this, however (about seven to fourteen days), after the second operation, they commenced to lose their hair, became slower and weaker in their movements, and developed an extraordinary postural attitude which was reminiscent of, but not identical with, illustrations of dogs suffering from artificial dietetic rickets, published in E. Mellanby's[56] articles. The animal appeared flat or splay-footed (Figure XI).

This remarkable hypotonia reached its most severe grade in an animal (P. 30) which, in addition to receiving the usual direct irradiation for five minutes, was three times subjected to further doses through the skin over the remaining kidney for a total period of twenty-one minutes, a two millimetre aluminium filter being used. By this means the posture was maintained for a period of

Figure XI.

This illustration is intended to show the muscular hypotonia referred to in the text. This animal was loath to stand for long and could not jump at all. It was fairly normal in other respects.

fourteen weeks (Chart I). A gradual return to normal occurred, and the dog resumed its usual activity. Accompanying and accentuating this phenomenon was a generalized wasting of all muscles, including those of the trunk and neck.

Figure XIa.

This illustration is intended to show the muscular hypotonia referred to in the text. This animal was loath to stand for long and could not jump at all. It was fairly normal in other respects.

Life in the few animals where renal failure apparently occurred, came to a sudden end usually about the eighteenth to the twenty-second day, the terminal illness usually not being longer than forty-

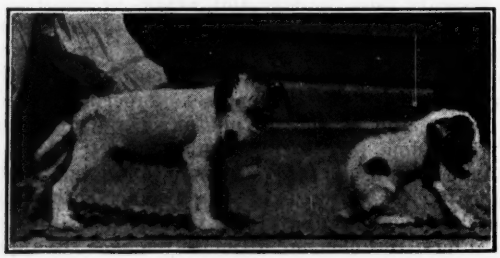

Figure XII.

Illustration of puppies, P. 7 and P. 9, taken a few hours before the death of P. 9, showing clearly their relative disproportion in size.

eight hours' duration. Fits were observed in three of these animals and œdema in two. The course and clinical features of this final illness represented an epitome of the sequence of events which began

in full-grown dogs twenty-one days after irradiation, with very rapid wasting, anorexia, drowsiness, dyspnœa and lethargy. Other animals, for example, P. 1, P. 2, P. 7, P. 30, P. 31, apparently survived this critical interval, and subsequently resumed their normal growth and activity.

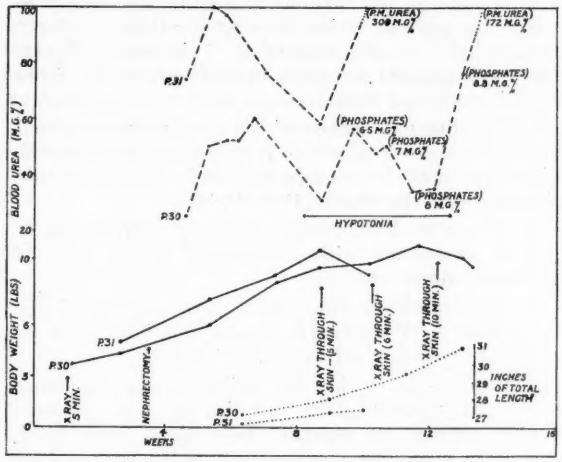

CHART I.

Bodily development and variations in blood chemistry in animals P. 30, P. 31 of Litter IX. Note similarity between curves and constant elevation (? non-renal) in blood phosphate without azotæmia. P. 31 was apparently the more susceptible, grew more slowly, and failed to survive a subsequent irradiation.

Effect on General Development and Growth of the Long Bones. Only fourteen animals are concerned where an opinion can be expressed as to their rate of growth. They can be grouped as follows:

(*a*) In only one case (P. 9) did this feature deserve the term "dwarfism".

This animal was one of a litter of four of about the same weight, born in the laboratory of known parents, and each received five minutes' radiation. One of the litter died from operative shock, while P. 9 survived for two weeks after nephrectomy. This animal not only ceased to grow further, but as a result of muscular wasting, seemed actually to shrink in size (Chart II). Death was apparently due to renal failure, precipitated by an intercurrent infection of the mouth. Figure XII illustrates this animal a few hours before death, taken with his brother (P. 7) who survived the same infection.

(*b*) Two further animals (P. 13, P. 29) lagged very distinctly behind other members of the same litter and thus showed a moderate degree of retardation.

The first of these lived about a month after radiation, which lasted five minutes, did well for a week after nephrectomy, then began to lose weight, although there was no suppuration or infection of any kind, and the animal died suddenly with tetany-like fits. Its weight after death was one pound less than at the second operation.

The other animal (P. 29) was the only one of the litter of four which received five minutes' dosage, to show any marked delay. Considerable blood was lost at the second operation, and only a quarter of a pound was added to the body weight in four weeks.

(*c*) One example (P. 7) will serve to illustrate the remaining six animals in which slight interference with growth occurred (P. 1, P. 10, P. 2, P. 16, P. 8, P. 24, P. 30).

This puppy was always slightly in advance of the remaining members of the litter and, apart from oral infection, seemed quite well after the second operation and appeared to grow rapidly. About a month after nephrectomy he seemed rather pot-bellied and exhibited a moderate degree of the hypotonia referred to above. This practically disappeared after a few days, but returned again in a more pronounced form about five weeks later, accompanied by distinct œdema in the face and limbs, some hard breathing and slightly yellow sclerotics. Two weeks later the animal seemed well again, but still displayed the same weakness in the legs. This slowly cleared up, however, in spite of two subsequent irradiations for a total period of thirty minutes through the skin over the remaining kidney. Nothing further developed until his death by accident six months after the original treatment. Chart II illustrates the above events graphically. During the last few months growth progressed at an average rate of one inch a month in total length.

The impression given by this and similar animals who survived for over four months (P. 1, P. 2, P. 7, P. 28, P. 30) was that any delay in growth which occurred was later wholly compensated for, so that these animals seemed likely to equal in size their parents.

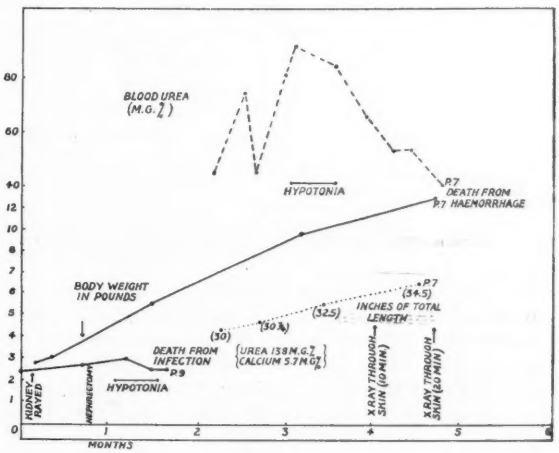

CHART II.

Illustrating opposite results occurring in two members of the same litter which received the same dosage of X rays. In P. 7 compensation has been so satisfactory that no disturbance followed two subsequent irradiations. (This should be compared with the changes in blood urea following the same treatment to P. 30, Chart I.) There appeared to be no reason for the sudden appearance of hypotonia three months after the initial treatment.

(*d*) Reference should be made to P. 1 and P. 2.

In these animals puberty occurred under the age of ten months and no serious interference with bodily development. Chart II illustrates the above events graphically. about twelve months of age. These two animals were already rather too advanced for treatment and were never embarrassed to any marked degree, as would be anticipated with a full-grown dog who had received less than a quarter of the standard dosage (Chart III). The results of repeated measurements of both the length of the radius, a process made simple in dogs in which marked eminences distinguish the extremities accurately, and of the circumference of the distal epiphysis of the same bone, failed to reveal any disproportion, at least within 0·5 centimetre. The time of appearance and character of the teeth were normal, and they failed to show any evidence of deficiency in lime.

The Radiograms. Certain changes could be observed almost invariably in all animals under

four months, irrespective of dosage and interval since treatment: (i) There were various degrees of an apparent increase of the normal lipping of the metaphysis seen in dogs (Figure XIII). (ii) There was some general rarefaction and thinning of the cortex of the shaft (Figure XIV). (iii) A uniform zone of haziness of varying depth close to the epiphyseal line was found. The bone appeared more dense and had a cotton wool or cloudy appearance in which all outlines of trabeculæ were lost. This usually commenced on one side of the metaphysis,

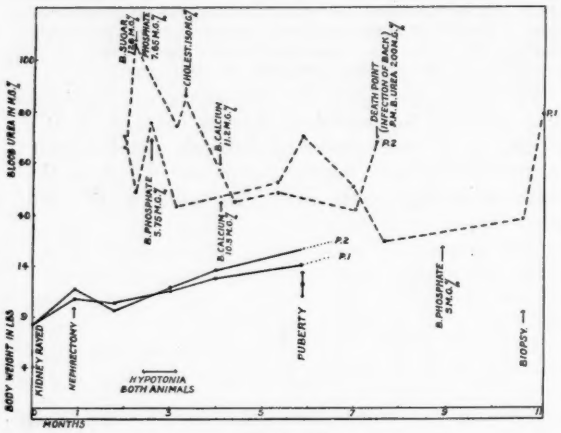

Chart III.

Illustrating two animals already half grown at time of irradiation. Compensation was attained two months after nephrectomy, during which time growth was delayed and hypotonia occurred.

then spread across and extended for as far as 1·25 centimetres (half an inch) into the shaft. Subsequently it became more dense and narrower, or disappeared slowly and completely. Figures XIII and XIV are intended to show these changes. (iv) In two cases bending of the shaft occurred, with buttress formation later, on the concave aspect (Figure XV). (v) Rarely a thin white line was to be seen in the shaft equidistant and parallel to the epiphyseal line, but separated from it by three to four millimetres. Such an appearance can sometimes be observed in malnourished infants.

The most pronounced disorganization of all was observed in the autopsy radiogram (Figure XVIII) of the most stunted of the series (P. 9), where the appearances suggested an extensive involvement with collapse, somewhat homologous to the late stages in the "woolly" human type. X ray examination at the time of nephrectomy revealed only a mild departure from normal.

Naked Eye and Histological Appearances of the Radiated Kidney.

The naked eye and histological appearances of the radiated kidney, together with the functional aspect of the question, are not discussed here, as it is intended to communicate these particular findings in more detail later, in conjunction with Dr. A. Bolliger. It is possible, however, to make the following provisional generalizations.

1. As in adult dogs, the period at which connective tissue strands begin to invade the damaged renal cortex (Figure XVII) is marked by a rapid clinical deterioration, often ending fatally, but in the smaller and more delicate kidneys of puppies, can result from five to ten minutes' standard irradiation and tends to occur during the twenty to thirty day period, rather than at the thirty to forty day period. In the case of P. 6, in which thirty-one days elapsed between operations, death from uræmia followed in four days after nephrectomy.

2. The unusual degree of hypotonia synchronized in the three cases in which it was most pronounced, with a distinct elevation in the level of the inorganic phosphate of the blood (P. 7, P. 30).

3. In one case only was arrest or retardation of growth associated with a lowering in the level of the total blood calcium.

4. In animals of more than four months of age five minutes of standard irradiation had little or no deteriorative effect on normal development.

5. Puppies below this age which survived for more than thirty days after irradiation soon became almost normal as regards their blood urea level, and grew normally.

Comparison of Lesion Produced with That Found in Renal Dwarfism.

The lesion histologically is more closely allied to that found in human renal dwarfism than was that found in adult dogs, but in no case were there produced the scarring, vascular involvement and round cell infiltration which are usually so characteristic of the disease in man, though human cases have been mentioned in which both nitrogen retention and cardiovascular hypertrophy are lacking.

Stunting of growth was, however, noted, but with the exception of one case was not so marked as to amount to arrest of development. This case, that of P. 9, has already been referred to, and although the urea retention amounted to less than 150 milligrammes, other possible causes of the dwarfism, such as infection, anorexia, were wholly terminal. Hence its remarkable arrest of growth was possibly by exclusion, directly due to the renal lesion, a type, therefore, of "acute renal dwarfism". No such delay occurred, however, in other animals dying more definitely of renal failure.

As regards the bone changes, no sign was found of epiphyseal enlargement or collapse, or of delay of growth in length of the long bones, although epiphyseal enlargement was not constantly present even in the human series, the reduced body weight apparently being the main adaptation to the diminished renal reserve.

Examinations of the radiograms showed an undoubted interference with calcium deposition on the shaft side of the epiphyseal line, but these appearances were definitely denied a renal origin, because their presence was well marked in the case of P. 1 before even the first operation. The presence of these changes so early in the experiments com-

pletely vitiated this method of controlling our results. In the case of P. 24, however, which died in all probability from renal insufficiency, the radiograms were strikingly free from any changes of any kind. The only remaining explanation is that of nutritional rickets, though, as stated above, every care was taken to avoid this fallacy, and long exposure to bright sunlight was given daily. Strangely enough, in the case of the most stunted animal (P. 9), macroscopical and microscopical examination of the epiphyses, which appeared most severely involved in the radiogram, even to the point of collapse, failed to reveal any disorganization, though the X ray appearances resemble those given by Park as characteristic of the "low calcium" type of rickets. Whether the atrophy and bending sometimes observed (Figures XIV and XV) were also of the same order, it is impossible to say. The blood changes as regards inorganic phosphate and calcium were certainly not those ordinarily seen in the rickets due to lack of vitamin *D*.

The question of the ætiology of the unusual posture illustrated in Figure XI is of interest. This hypotonia, though possibly of nutritional origin, was of a more severe grade than was seen in experimental nutritional rickets (Mellanby) and coincided with periods of elevated blood urea. In view of the known intimate relations existing between phosphate metabolism and muscular contraction, it is worthy of note that the change appeared while the phosphate level of the blood was also raised, but Bolliger has not noted any such phenomenon following intravenous injections of phosphates in adult dogs.[57]

Regeneration and early functional stability defeated one's object in the older virile animals, while younger material was so decimated by intercurrent accidents, to which a renal impairment rendered them more susceptible, that it was difficult to maintain the slow chronicity and cause the gradually increasing renal failure which favours the appearance of the human disease.

Conclusions and Summary of These Experiments.

1. Following the technique of Bolliger, Laidley, and Earlam, a series of thirty puppies of age from one and a half to four months were subjected to five to ten minutes' standardized irradiation of one kidney, followed after an interval by hetero-lateral nephrectomy, in an attempt to produce an experimental renal dwarfism which could be of use for pathological and therapeutic research in the subject.

2. Histological examination of the radiated kidney up to seven months after treatment revealed a glomerulo-tubular nephritis of an extensive character, which differed somewhat from the appearance seen in adult dogs. The growing organism in several instances showed itself to be possessed of remarkable recuperative powers as regards this type of renal lesion.

3. The lesion produced in this way, though azotæmic, is not strictly comparable to that usually seen in human interstitial nephritis, and no arrest of growth occurred which could be termed an indubitable "renal dwarfism". Mild radiographic evidences of rickets of probable nutritional origin were observed, and several animals developed an extraordinary degree of muscular hypotonia during periods of azotæmia and elevated blood phosphate.

Summary and General Conclusions.

1. A review of the literature and recorded cases of renal dwarfism is presented, including three fresh examples of the disease, one of which is apparently the severest on record.

2. Suggestions are offered towards the ultimate ætiology, based on the conception of a pre-natal factor interfering with the full development of the kidney. The suggestion is made that certain cases in younger patients may represent a perpetuation of an original nutritional rickets, following the establishment in early life of a severe grade of renal insufficiency. In a small series of cases no evidence of lasting damage to the child following toxæmias of pregnancy has been found.

3. A full description is made of the clinical, pathological, radiological and diagnostic features of the disease. Emphasis is laid on the necessity for thorough renal testing before undertaking the operative cure of any case of *genu valgum*.

4. The micropathology is compared with that of other types of chronic nephritis in the growing organism, without distinguishing one feature peculiar to dwarfism.

5. The accepted conception of the mode of interference with bone growth is related, and an explanation given of the differences of a biochemical order existing between renal dwarfism and adult nephritis.

6. Certain facts emerge from this review which should receive consideration in the prognosis of a case of juvenile interstitial nephritis, since the occurrence of headache, marked impairment of urea excretion, and fits should not be viewed with quite the same gravity as in adult life. The appearance of cardiovascular hypertrophy in this connexion seems to be of no value and, if it occurs at all, is dependent on renal factors quite dissociated from those responsible for the bony changes.

7. An account is given of an experimental attempt to reproduce the disorder.

Acknowledgements.

I gratefully acknowledge my indebtedness to Dr. D. H. Paterson and Dr. L. Parsons for extending my interest in the subject and for permitting me to refer to their case records; to Dr. A. McIntosh, Dr. R. Sear and Dr. L. Dey for allowing me to comment on patients admitted under their care; to the late R. Gordon Craig and Dr. A. H. Tebbutt for laboratory facilities; to Dr. H. C. E. Donovan for permission to examine records at the Hospital for Women, Crown Street, Sydney; to Dr. Cronin and the staffs of the Departments of Radiology and Urology, Royal Prince Alfred Hospital, for technical assistance; and especially to Dr. A. Bolliger,

whose assistance in the experimental work was indispensable and from whom, with Dr. Keith Inglis and Dr. M. Earlam, I have received helpful criticism. Finally, I wish to thank the Senate of the University of Sydney for a small but adequate grant from the Hebden Memorial Fund.

The specimens from Case I have been presented to the museum of the Sydney Medical School.

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Reports of Cases.

CARDIAC FAILURE TREATED BY "CORAMINE".

By A. M. WATKINS, M.B., B.S. (Melbourne),
Roseville, Sydney.

I WAS summoned urgently to the bedside of A.B., aged sixty-eight years, as he was apparently dying from a severe heart seizure. At the time of my arrival the patient was unconscious. His cheeks were hollow and

his eyes sunken, his forehead was covered with a clammy sweat, and his colour was ashen. There was an audible "death rattle" in the chest. The chest, both anteriorly and posteriorly, was full of mucous râles. I did not think it would be possible to resuscitate this apparently moribund man.

Two vials of "Coramine" were at once injected hypodermically, and within ten minutes the "rattle" in the throat had disappeared, together with the clammy sweat, and consciousness was restored. A second hypodermic injection of 16 milligrammes (one-quarter of a grain) of morphine, 1·08 milligrammes (one-sixtieth of a grain) of strychnine, and 0·65 milligramme (one one-hundredth of a grain) of atropine, was then given. This was followed by a dose of aromatic ammonia and compound spirit of ether. A large amount of flatus was expelled from the stomach, and the patient stated he felt much better.

Some six hours afterwards, a great deal of blood-stained mucus was expectorated. Tincture of digitalis was given in a dose of 0·6 cubic centimetre (ten minims) every four hours. Two days later the hæmoptysis had subsided, the patient was able to sit up in an arm-chair, and his heart was functioning efficiently.

I am of the opinion that the use of "Coramine" was the essential factor in saving this man's life; it acted as a most powerful cardiac and respiratory restorative, allowing the exhibition of morphine without the fear of its characteristic effect on the respiratory mechanism.

FRACTURE OF THE CRICOID CARTILAGE.

By James H. Younkman, M.B., B.S. (Adelaide),
Wagin, Western Australia.

A.H.H., aged twenty-six years, was standing on the off side of his horse on December 4, 1931, at about 4 p.m., when he sustained a kick on the left side of his neck. On recovering from the initial pain and dyspnœa produced by the injury he rode five miles on horseback to his nearest neighbour, who took him by motor car thirty-five miles to Lake Grace, where he was seen by Dr. W. B. Fry.

At this time there was a small amount of subcutaneous emphysema in the lower half of the neck anteriorly, and air could be felt to leave the larynx and enter the subcutaneous tissues of the neck with each respiration. The patient was sent into Wagin (another eighty miles) and was admitted to the Wagin District Hospital at 1.30 a.m. on December 5.

On examination he was standing at the foot of his bed, grasping the rail and using his accessory muscles of respiration. There was intense dyspnœa with inspiratory and expiratory stridor, the former being much worse. Cyanosis of a deep blue tint was present and the patient could only whisper a word or two at a time, this being accompanied by much pain. There was subcutaneous emphysema of the forehead, face (both eyes were closed), neck anteriorly and posteriorly, chest, abdomen and back as far as the hips, right arm to the wrist, and left arm to the fingers.

Under chloroform anæsthesia an attempt was made to pass an intratracheal catheter. This failed owing to the presence of a large amount of submucous emphysema of the larynx. In palpating the suprasternal notch prior to incision the tissues pitted on pressure, leaving a hole about the size of a halfpenny. With each breath a bubble of air could be seen to enter this hole, three breaths being sufficient to obliterate it. Probably at this time more air was entering the subcutaneous tissues than the lungs. Tracheotomy was then performed, with immediate relief of cyanosis and dyspnœa.

Convalescence was rapid, but was accompanied by slight cough, expectoration of blood-stained sputum, and evening pyrexia. The tube was removed on the eighth day. The emphysema disappeared in three weeks. As the voice was still husky, I referred the patient to Dr. H. J. Gray, to whom I am indebted for the following report, dated January 8, 1932.

The present position is that the right side of the larynx is quite all right. There is only very slight movement of the left cord, only sufficient to make me satisfied that the recurrent laryngeal nerve is functioning. The fracture appears to have involved the aryteno-cricoid joint. I cannot see that his thyreoid cartilage was fractured, which is a more common injury. There was probably a lesion of the lining membrane below the cord to allow the emphysema to occur. I consider it quite possible for some further return of movement, but cannot suggest any line of treatment to assist this desirable end.

FUNCTIONAL ANOMALY OF THE TONGUE.

By Henry Shannon, M.D. (London),
Honorary Physician, Austin Hospital, Heidelberg, Victoria.

The accompanying illustration shows a rather uncommon agility of the tongue, and as I have never seen it mentioned or portrayed in print, I thought it worth while to do so.

The tongue itself was not above average length, and the young man (a trained singer) to whom it belonged, could with great ease, upon opening his mouth, turn the organ backwards and insinuate it into the naso-pharynx above the soft palate. This unwonted intrusion may not have been without pathological significance, for he has suffered with an inflammation of the right antrum and much nasal catarrh.

Reviews.

THE DIAGNOSIS OF NERVOUS DISEASES.

The work on the diagnosis of nervous diseases by Sir James Purves Stewart first appeared twenty-five years

ago, and to find it now published in a seventh edition is in itself sufficient tribute to its popularity.[1] The book is so well known to all interested in neurology that to them it is only necessary to announce the appearance of the new edition, and to say that it is the introductory chapters on anatomy and physiology and those dealing with postural reflexes, intracranial tumours and disorders of sleep, which have been specially revised. To new readers, however, it may be indicated that the subject matter is designedly arranged to enable an observer in a diagnostic quandary to take, analyse, and piece together to a complete whole, signs and symptoms which he may see. To meet this purpose, the book is divided into chapters each of which deals with a special class of sign, symptom, or disorder of function, such as delirium, fits, disorders of articulation, pain, paralysis, postures, and gaits, to name the more important. These in all their variations are described in detail. Thus, in the matter of paralysis, we are told how to recognize and where to locate the lesion in the various paralyses of upper neurone type, and how to differentiate them from functional paralyses. Then we are given the characters of paralyses of lower neurone type, exemplified by infantile paralysis, polyneuritis, and lesions of peripheral nerves, and, lastly, recurrent and transient palsies, illustrated by *myasthenia gravis*, Thomsen's disease and intermittent claudication. It must not be supposed that the book is a mere compendium; on the contrary, the material is openly displayed and abundantly illustrated, and withal put before the reader in attractive form by one who is known as a great demonstrator as well as a master of neurological diagnosis.

Neurology in its clinical bearings alone is a big subject; year by year fresh contributions pour in and old conceptions go by the board; and it is only because we realize this and the difficulty of collecting all that is written, that we venture to indicate certain passages which might be altered. For one thing, experimental physiologists now agree that changes in plastic tone following sympathectomy are due, not, as Hunter and Royle originally thought, to severance of a supposed connexion between sympathetic nerve and muscle, but, as Wilkinson has shown, to withdrawal of sympathetic influence upon blood vessels. Also in connexion with the problem of precocious sexuality, it might be well to mention that more and more cases of this affection come to be recorded, in which there is a brain lesion, usually a tumour, and a perfectly normal pineal gland. And we would welcome relief from our confusion by a clearer exposition of the clinical distinctions between chronic anterior poliomyelitis, progressive muscular atrophy and amyotrophic lateral sclerosis.

These, however, are minor considerations and our final comment is that this book continues to stand alone in its eminence as a guide to neurological diagnosis.

THE AUSTRALIAN ABORIGINAL.

A BOOK which should be of interest to every Australian has been written by Stanley D. Porteus, Professor of Clinical Psychology in the University of Hawaii. He has called it "The Psychology of a Primitive People: A Study of the Australian Aborigine".[2] The study reported by Professor Porteus was undertaken at the request of the National Research Council of Australia, and was conducted with the cooperation of the University of Hawaii and by the aid of a grant from the Rockefeller Foundation.

In his preface the author states that, prior to this work, with the exception of one or two very minor studies involving small groups of half-castes as well as full-bloods, nothing whatever had been attempted in the way of psychological examinations of individuals of the aboriginal race. He refers to his work as a "pioneer study" and to the field as a "virgin field in science, offering the promise of exceptional returns for labour expended in it". The statements are somewhat misleading. A great deal of work has been done on the psychology of the Australian aboriginal; Professor Porteus refers throughout his book to the writings of others, and these references make his work more interesting than it otherwise would be. If the "pioneer work" may be taken to refer to the application of so-called psychological tests to a series of persons, the statement may be allowed to stand. Further, if this statement stands, Professor Porteus must surely feel disappointed, for his "exceptional returns" are lacking. The results of his application of certain tests to aborigines do not lead him much, if any, further than investigation without tests has led others. In spite of this, it cannot be held that the book is not full of interest. Those who have not studied the Australian aboriginal will find it most informative, if a little long-winded, and will certainly wish to pursue the study further.

The book is divided into two parts. In the first part the author describes the "physical background of the aborigines" and gives details of his tour in north-west and central Australia. In the second part he deals with such questions as social organization, totemism, ceremonial observances and initiation, and gives details of the results of his psychological testing. A great many of his observations were made on aborigines who had been in contact with the white man and even under his instruction for some considerable time. He points out that the aboriginal (he uses "aborigine" for the singular of aborigines, which is incorrect) will more often than not deceive the white man who is looking for information on native customs, and at the same time he is quite certain that he himself has not been "spoofed". Perhaps he was not. In any case his descriptions are most interesting. He shows, as others have done, that the native has adapted himself to his environment in a surprising fashion. He describes the search for food, which occupies the whole of the aboriginal's time. His account of the ceremonies of initiation carried out on all males would strike terror into the heart of the average white man. The account of the primitive beliefs and customs of the people, of the marriage laws and of the usages in regard to sex show that the aboriginal is not lacking in intelligence or foresight. It may be noted in passing that when Professor Porteus enters the realms of theology he loses himself. He does not know the difference between the doctrine of the virgin birth of Christ and that of the immaculate conception; in his reference to the immaculate conception on page 221 he obviously means the virgin birth.

There is an interesting chapter on the origin and dispersion of the aborigines. It is impossible in a short review to do justice to this question. It must suffice to state that the author accepts the view advanced by Eyre that the original landing place was in the north-west and that dispersion took place from this point. He looks on north Queensland as too fertile an area for the original landing place; if natives landed here, how and why, he asks, should they migrate to the inhospitable areas of the desert in the west?

The tests carried out by the author included physical measurements, psycho-physical tests, such as grip tests, back and leg lift, and vital capacity estimation. The intelligence tests included the Porteus maze test, the Porteus maze and assembling test, the xylophone test, form board test, digits memory test, dot test *et cetera*, and a drawing test for children. The greatest number of adults subjected to any one test was 120, and the children numbered 147. Doubt may well be expressed as to the value of these tests, applied in the way the author applied them, as an indication of the intelligence of the aborigines.

From the scientific point of view Professor Porteus's book may be somewhat disappointing, but he has certainly written something which should be read by all Australians who know but little of their country or of the native races to which it originally belonged.

[1] "The Diagnosis of Nervous Diseases", by Sir James Purves Stewart, K.C.M.G., C.B., M.D., F.R.C.P.; Seventh Edition; 1931. London: Edward Arnold. Royal 8vo., pp. 738, with illustrations. Price: 35s. net.

[2] "The Psychology of a Primitive People: A Study of the Australian Aborigine", by S. D. Porteus; 1931. London: Edward Arnold and Company. Demy 8vo., pp. 453, with illustrations.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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THE MEDICAL SERVICE OF THE ROYAL AUSTRALIAN NAVY.

FOR some years past much difficulty has been experienced in Great Britain in obtaining suitable recruits for the medical branch of the Royal Navy. The difficulty has been so great that concern has been expressed by more than one naval authority. Surgeon Rear-Admiral C. Marsh Beadnell, R.N. (retired), writing on "Naval Medical Service as a Profession", quoted remarks by the First Lord of the Admiralty in his speech in Parliament on March 17, 1931, on the naval estimates. The First Lord said:

> I regret that some concern is felt by my advisers over the difficulty of obtaining an adequate supply of medical officers in spite, may I say, of special efforts which have been made during the last twelve months by the Medical Director-General.

Surgeon Rear-Admiral Marsh Beadnell, who, be it noted, had retired from the service and could therefore speak his mind, commented on the matter in the following words:

> If you do not get the best type, you must obviously get a mediocre or inferior type, unless, indeed, as has already happened, you get no type at all, any one of which eventualities must react on the whole Navy. You cannot in fairness expect an A1 health bill in return for C3 attention.

The last sentence in this statement lies at the root of the matter.

The troubles of the Royal Navy have been reflected in the Royal Australian Navy, and disabilities peculiar to Australia have arisen. These require serious and immediate attention. The Federal Committee of the British Medical Association in Australia had before it at its last meeting in February, 1932, a report from Surgeon-Commander W. E. Roberts, R.A.N. (retired). In this report Surgeon-Commander Roberts enumerated the disabilities under which medical officers of the Royal Australian Navy are called upon to work. The difficulties and disabilities are real, and the official ear is so deaf that publicity must be given to the facts.

In the first place, the pay of medical officers is not adequate. The rate of pay for medical officers in the Royal Australian Navy is below that of the Royal Navy, except in two instances in which the Australian pay is in excess by one penny. Those who contend that naval medical officers are well paid, lose sight of the cost of medical education and of the late age at which medical officers enter the navy. The cost of a medical education at a low estimate is somewhere in the neighbourhood of £1,500, five or six years are spent in a medical school, and a graduate enters the navy at twenty-five or twenty-six years of age. Most non-medical officers of the navy enter the service at an early age, and to their education and training a large amount of public money is devoted. In the Royal Navy an allowance of "charge pay" is made. This is an allowance made to medical officers in charge of hospitals, sick quarters and hospital ships. The rejection of charge pay for medical officers in the Royal Australian Navy is one of the greatest anomalies of the service. Charge officers in the Royal Australian Navy have the same responsibilities as charge officers in the Royal Navy, and they have to do much special work of inspection, instruction and supervision which in the Royal Navy is carried out by special officers. The uniform allowance of £50 is inadequate. Allowances are being curtailed without any regard to the effect that service requirements have on active pay. In England much attention was focused on this question and definite proposals were made by the British Medical Association (see the Supplement to *The*

British Medical Journal, April 25, 1931, page 152). The amount recommended in England was £100. The £50 allowance in Australia has been reduced by 20%. The matter of deferred pay of medical officers in the Royal Australian Navy has always been a bone of contention and has been discussed in this journal on previous occasions. The treatment meted out to medical officers demonstrates the official view that deferred pay is money withheld from an officer for the purpose of liquidating liabilities that might have been incurred during the period of his service. Deferred pay is apparently not intended to serve as a basis for purchasing an annuity. So much for the financial side.

In regard to medical work and administration Dr. Roberts has much to report. Hospital and post-graduate courses have been curtailed. The qualifying courses for promotion to surgeon-commander have been postponed almost indefinitely. This is nothing less than a breach of faith to men who, by length of service, have shown that they regard the naval service as their life's work. If a man has no opportunities of promotion, and if no facilities are provided for post-graduate study, knowledge will suffer, interest will wane and the service become inferior. Other conditions, irksome and sometimes intolerable, include the curtailment of the powers of medical officers. There is interference in medical matters by non-medical members of the civil service. No rank higher than surgeon-commander is attainable in the Royal Australian Navy—no provision is made on the pay list for pay higher than that of surgeon-commander. Finally, the position of the Director of Naval Medical Services is anomalous. He is not allowed to be present at meetings of the Naval Board when medical matters are discussed. The rank of the Director of Naval Medical Services is equivalent to that of surgeon-captain, but the pay is that of his substantive rank, that of surgeon-commander. Surgeon-commanders under him sometimes, by reason of certain allowances, receive higher pay than he; such a condition does not exist in any branch of the Royal Navy.

From this dismal recital it is clear that the Naval Medical Service in Australia holds out no attraction to medical graduates who are looking for a career. Those in the service can do little to remedy the unsatisfactory state of affairs. Officials in charge of government departments lose sight of the rights of individuals, and they forget that neglect of the individual has a detrimental effect on the efficiency of the service. Even if financial considerations of the moment make it impossible to adjust the anomalies in the regular pay of naval medical officers, some attention should be paid to the other matters mentioned, especially to the question of status and to the provision of facilities for post-graduate work. Unless something is done, medical graduates will be well advised to avoid service with the navy.

Current Comment.

THE EXCRETION OF "NOVARSENOBILLON".

"Salvarsan" and its various modifications are widely used throughout the world in the treatment of many conditions in addition to syphilis. In tropical countries especially they are employed freely, often by unqualified assistants. Injections are often lightly made, and it is doubtful whether the majority of persons give more than an occasional thought to the ultimate fate of the injected material. And yet it is very important that the medical practitioner should know what chemical changes take place, whether the drug is stored in the body, and what is the rate of its excretion. Fixation of a drug in tissues or organs suggests that there is a danger of a cumulative effect and of consequent poisoning. Obviously, if the rate of excretion is slow, poisonous effects are more likely than if it is rapid. These factors are of great importance also from the point of view of therapeutics. The medical practitioner should ask whether the arsenobenzol compound is of therapeutic value only as such, or whether its activity is due to liberated arsenic, and whether its effect is immediate and transient, or prolonged by virtue of arsenic stored in the body.

The clinical significance of the excretion of "Novarsenobillon" in the urine of syphilitic patients is discussed by V. E. Lloyd and N. L. Lloyd in a recent interesting paper.[1]

Their test for the presence of "Novarsenobillon" (or other trivalent arsenobenzol compound) in the urine is briefly as follows: To five cubic centimetres of urine 0·5 cubic centimetre of decinormal hydrochloric acid and one cubic centimetre of a 0·5% solution of sodium nitrite are added separately in that order. In a second test tube are placed one cubic centimetre of a saturated solution of resorcin and a similar quantity of a 10% solution of sodium hydroxide. After the contents of the first tube have

[1] *Guy's Hosptial Reports*, October, 1931.

stood for ten minutes they are added to the second tube. If "Novarsenobillon" is present, a red colour develops. This is compared with standard solutions, and a rough quantitative estimation is thereby made. They point out that the urine of persons who have recently taken phenacetin, reacts in a similar manner. In all, they made 3,892 tests on the urine of 302 male patients.

They regarded excretion as normal when there was a definite reaction within the first three hours of the administration of "Novarsenobillon". When there was no evidence of "Novarsenobillon" in the urine within three hours of the injection, they considered that the rate of excretion was subnormal.

Their investigations revealed that neither the stage of the disease (primary, secondary or latent) nor the age of the patient had any apparent influence on the rate of excretion of "Novarsenobillon" in the urine. They found that in some instances the rate of excretion remained constant throughout the period of treatment, in some it increased, in some it decreased, and in some it was irregular. They point out that other investigators have noted a variability in the rate of elimination of organic arsenic.

The excretion rate of 103 patients whose urine contained no abnormal constituents, was normal in 32 instances, subnormal in 65, doubtful in six. The excretion rate of five patients whose urine contained albumin, was normal in three and subnormal in two instances. These workers admit that this information is scanty, but remark that it does not reveal any indication that the rate of excretion is influenced by abnormal renal conditions. Bismuth is known to have a deleterious effect on the kidneys; but there was no evidence that bismuth therapy caused a decrease in the rate of excretion. It should not be concluded from these results that renal efficiency is of no importance in the elimination of "Novarsenobillon". It has not been clearly proved in these investigations that the rate of elimination is ever independent of renal efficiency.

Of 58 patients whose rate of excretion was normal, only 18 were intolerant of "Novarsenobillon", while of 89 whose rate of excretion was slow, 48 were intolerant. It is unfortunate that the investigators do not mention on what symptoms they based their diagnosis of intolerance. The incidence of toxic jaundice did not appear to be influenced by the rate of excretion. The authors point out that, as shown by Voegtlin, Dyer and Miller, ligation of the bile duct slightly increases the liability to poisoning by arsenobenzol compounds. They suggest that as far as the liver's tolerance is concerned, elimination in the bile is a more important factor than elimination in the urine.

Voegtlin and Thompson, employing arsenical compounds in the treatment of rat trypanosomiasis, showed that the therapeutic effect was greater when arsenic was more slowly excreted. They regarded the temporary retention of the drug in the tissues as essential to efficient trypanocidal action.

Employing the Wassermann test as a guide to results, V. E. and N. L. Lloyd attempted to ascertain whether the therapeutic effect of "Novarsenobillon" was greater in their cases when the rate of excretion was subnormal. Their results were the reverse of what might have been expected in view of the work of Voegtlin and Thompson, but the numbers were small, and conclusions could not logically be drawn. It should be noted also that Voegtlin and Thompson made estimations of the quantity of organic arsenic excreted in both urine and fæces, whereas V. E. and N. L. Lloyd examined the urine only, employing a test that did not permit the making of accurate quantitative estimations. It is probable that the greater part of the organic arsenic administered in "Salvarsan" and similar drugs is excreted in the fæces.

It is questionable whether a tardy commencement of excretion can be invariably taken to signify a slow rate of excretion. The work of V. E. and N. L. Lloyd is of very great interest, but the conclusions they draw from it should not be accepted without further investigation. More accurate quantitative estimations of the organic arsenic excreted in urine and fæces must be made over a period of months. This would be a laborious task, but it is only by some such means that definite knowledge concerning the storage of arsenobenzol compounds and the rate of their excretion can be obtained. By means of ordinary tests the presence of arsenic in the urine may be revealed eight or ten days after an intravenous injection of "Salvarsan"; if more delicate tests are employed, traces of arsenic can be found months after an injection. "Novarsenobillon" and similar preparations are excreted much more rapidly. Willcox found no abnormal accumulation of arsenic in the liver of persons who had died of toxic jaundice. Other observers have found that arsenic is stored in the rabbit's liver after the administration of arsenobenzol compounds, and that the period of storage is prolonged by each succeeding injection. Obviously, a great deal of work is required, if only in the correlation of these apparently anomalous results.

MEMORIAL TO ROBERT HENRY TODD.

Members of the British Medical Association throughout Australia are reminded that the Council of the New South Wales Branch has decided to perpetuate the memory of the late Robert Henry Todd by the establishment of an annual prize at the University of Sydney in medical jurisprudence. Robert Henry Todd was lecturer in medical jurisprudence for twenty years. He served the Branch of the British Medical Association in Australia in various ways. Doubtless many medical practitioners will welcome an opportunity of showing their appreciation of his services, and his old students of paying a tribute to him. Subscriptions may be sent to the Medical Secretary of the New South Wales Branch, 135, Macquarie Street, Sydney.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Treatment of Pelvic Inflammation.

B. ZONDEK (*Deutsche Medizinische Wochenschrift*, October 30, 1931) discusses the use of the anterior pituitary hormone, "Prolan", in the treatment of pelvic inflammation. He has noted at operations, when "Prolan" has been previously used, that the pelvic organs were definitely hyperæmic and turgescent, similar to those of the pregnant state. The results of 140 cases of acute and chronic tubo-ovarian inflammation are given. When pus is suspected, aspiration with a needle through the posterior fornix is always first performed, and this is followed in two or three days, whether there be a rise of temperature or not, by injections of "Prolan"; 100 rat units are given intramuscularly every day for ten to twelve days. The injections are stopped if uterine hæmorrhage or a marked reaction occurs. The cessation of pain is very characteristic. This line of treatment is particularly useful in cases of abscess in the pouch of Douglas. Shortening of the period required for treatment and convalescence was noted by Zondek. He considers that the combined use of puncture and injections of "Prolan" marks an important advance in the treatment of chronic inflammatory lesions of the female genitalia and in particular eliminates many unnecessary mutilating operations.

Friedman Modification of the Aschheim-Zondek Test.

M. DAVIES AND E. W. WALKER (*The New England Journal of Medicine*, September 17, 1931) report the result of the Aschheim-Zondek test performed on sixty-five patients. They discuss briefly the original test, the reliability of which has been established, the accuracy in 5,000 cases reported in the literature having been 98%. The technique of the Friedman modification (in which rabbits are used instead of mice) and the methods for the proper selection of the animals fit for the test are described in Friedman and Lapham's own words. The Friedman method as modified by Schneider and as advised by the latter in a personal communication to the authors, was used by them. A single injection of ten cubic centimetres of a voided first morning specimen of urine was used. Instead of killing the animal at the end of forty-eight hours, it was opened at the end of twenty-four to thirty-six hours and the ovaries were examined. It was then closed again and was available for subsequent tests. In one case an animal opened at the end of twenty hours was found to be normal. It died during the night, and on being reexamined in the morning was found to manifest a positive reaction. Several apparently normal animals were reoperated on at the end of forty-eight hours, but, except in one case, no change was found in the result. The authors have not, as yet, made any further use of any of the rabbits which gave positive results, but intend doing so. They have not yet found any specimen sufficiently toxic to kill the rabbit. They have not used any preservative in the urine, but have insisted that all specimens be collected in sterile bottles, and have filtered all the urine through a Berkefeld filter. The test is quite as delicate as the original Aschheim-Zondek test. The test fails to give a reaction in most cases within forty-eight hours after delivery. It is thus a good method for determining the death of the ovum. Detailed statistics are given to demonstrate the value of the method.

Ovarian Function after Hysterectomy.

H. SEIDENTOPF (*Monatsschrift für Geburtshülfe und Gynäkologie*, January, 1932) has analysed a considerable number of cases of hysterectomy to determine whether retention of the ovaries is important. When both ovaries were retained, the patients experienced regular premenstrual sensations for a considerable period, thus showing that the menstrual cycle was maintained. When dysmenorrhœa was a prominent symptom, these sensations were sometimes rather painful. When one ovary had been left *in situ*, the clinical picture was similar. In only one instance was cystic degeneration with cessation of all sensations noted. In this case it was impossible to determine whether this was due to mechanical or endocrinal causes. When both ovaries were removed, definite climacteric symptoms were induced. The continuance of ovarian activity was also corroborated in a number of cases by investigation of blood sugar curves. The author concludes that all the evidence points to the value of preserving as much as possible of ovarian tissue when the uterus is removed.

Genital Symptoms Associated with Cerebral Syphilis.

R. BRUHL (*Monatsschrift für Geburtshülfe und Gynäkologie*, November, 1931) describes a case of severe menorrhagia which was uninfluenced by local treatment and in which no uterine cause could be discovered. General examination revealed the presence of *diabetes insipidus*, acromegaly and excessive fat deposits indicating disturbed metabolic changes. X ray examination of the *sella turcica* region disclosed no abnormality, the fundi were normal, as was also the cerebro-spinal fluid. The blood gave a strongly positive reaction to the Wassermann test, and the cerebro-spinal fluid failed to yield a reaction. Cerebral syphilis was diagnosed, and immediate improvement was noted following specific treatment. The author has collected twenty-five cases from the literature. In ten instances in which an autopsy was performed, gummata of the pituitary gland were noted, and in some interstitial inflammation and scarring of the connective tissue.

Pregnancy and Carcinoma.

S. BÍRO (*Monatsschrift für Geburtshülfe und Gynäkologie*, November, 1931) discusses the occurrence of carcinoma of the cervix and pregnancy. The details of eleven cases are given. The radical operation was performed in all instances. One patient died after operation, and two later from recurrences. The author maintains that in operable cases radical measures should be adopted. The vaginal route should be used if the patient be seen in the early months of pregnancy, and the abdominal route for more advanced cases. Occasionally, in the interests of the fœtus, the operation may be postponed for a few weeks. In inoperable cases, if the patient is seen early in pregnancy, supravaginal hysterectomy should be performed at once. When pregnancy is further advanced, Cæsarean hysterectomy is performed when the fœtus is viable. In all cases radium and deep X ray therapy should be used to complete the treatment.

Myoma of the Vagina.

W. SCHILLING (*Monatsschrift für Geburtshülfe und Gynäkologie*, November, 1931) describes a case of primary myoma of the vagina and summarizes the literature on the subject. The majority of such tumours are fibromyomata. In shape most are round or else conform to the outlines of the vagina. The mucous membrane covering the tumour remains intact, unless mechanical influences destroy it. The majority appear to be of the submucous type and are hard in consistence. Torsion of the pedicle is common. The anterior vaginal wall is the common site of growth. They mainly occur in the thirty to forty-five age group and cause few symptoms beyond those of pressure on neighbouring organs. Whilst ulceration has been frequently noted, no cases of sarcomatous degeneration are on record. The usual treatment is enucleation and ligature of the pedicle.

Diagnosis of Ectopic Pregnancy.

H. LEWIN (*Monatsschrift für Geburtshülfe und Gynäkologie*, October, 1931) considers that in the majority of cases the diagnosis of ruptured ectopic pregnancy is not difficult. However, in unruptured cases mistakes may often be made, especially in the presence of ovarian cysts, tubo-ovarian swellings, or when the appendix is adherent to the appendages. A positive result to the Aschheim-Zondek test is of value in unruptured ectopic pregnancy. He also stresses the value of the leucocyte count, the sedimentation test and puncture of the posterior fornix. Despite these aids to diagnosis, difficulties often occur, and the detailed histories of four cases are given in which the real causes of the symptoms

were: ovarian cyst with omental adhesions, spontaneous rupture of the pregnant uterus through an old scar, appendicitis, a very young tubal pregnancy which had apparently eroded into an artery in the tubal wall.

OBSTETRICS.

Maternal Mortality and Morbidity in Germany.

MAX HIRSCH (*Münchener Medizinische Wochenschrift*, May 22, 1931) states the position of the mother and child in Germany and pleads for an improvement in the risks of childbirth. In contrast to the advance in surgery, and in spite of improvement in antisepsis and asepsis, the increased supply of medicaments in the obstetrical cupboard and improved hospitals and institutions, there has been practically no progress in obstetrics during the past ten years. The reasons the author gives for the maternal mortality are twofold: First, there has been no change for several decades in the obstetrical organization. Obstetrical operations of more and more difficulty are carried out today in the private house under the most unfavourable conditions, contrary to all claims of surgical asepsis. Secondly, there is far too much internal (vaginal) examination in the private house. Certainly the introduction of rubber gloves has diminished the dangers of this procedure, but a sterile glove can still carry organisms from vagina to uterus. Thus the author explains the yearly loss of 3,000 mothers from puerperal infection. In 1928 6,532 women died as a result of either confinement or abortion. Unfortunately the statistics do not separate these, and a correct idea of the proportion cannot be gained. In addition, from the same conditions arose 12,000 to 13,000 cases of the severe types of puerperal infection which were not fatal. These figures accrue from 1,500,000 births. In the State of Prussia in 1928 there were 2,408 deaths following confinement and abortion, but the author points out how incomplete this figure is, because of the failure of notification of many deaths in that State under the heading of puerperal cause. The improvement resulting from hospital treatment in contrast to that in the private house is estimated as five times less as far as risk of puerperal infection goes, and ten times less in the mortality from such infection. The author suggests that two essential requirements must be: (i) Transference of operative obstetrics from private house to hospital; (ii) the training of the obstetrician from the antiseptic to aseptic. Women suspect of a difficult confinement should arrive in hospital, as far as possible, at the right time and free from infection. The author further pleads for the cultivation of an external method of examination of the woman, with a corresponding diminution of vaginal examination. When external investigation fails, rectal examination should be practised before vaginal. Internal measurement of the pelvis he denounces as an additional source of infection, which, at the same time, is unnecessary. Delayed birth (over thirty hours in a *primipara* and over twenty in a *multipara*) should be reported by the midwife to a medical practitioner and the patient should be transferred to the hospital. From the side of the infant the author deals with *asphyxia neonatorum* as in the majority of cases due to a prolonged labour. In this connexion he recommends an hourly record of the fœtal heart sounds. The basis of a correction of the maternal mortality and morbidity lies, he asserts, in obstetrical organization.

Post Partum Hæmorrhage.

H. KÜSTNER (*Deutsche Medizinische Wochenschrift*, August 21, 1931) discusses the causation of atonic bleeding after the delivery of the placenta. In many cases hæmorrhage occurs despite an apparently normal third stage, with placenta and membranes complete and no signs of any traumatic lesions of the genitalia. A careful investigation of the uterine cavity shows the formation of a firm clot in the region of the placental site acting as a foreign body, preventing proper uterine contraction and retraction. This clot formation normally occurs, but in some instances, from unknown causes, it forms a larger mass, which is the cause of the continual leaking of blood. The loss is generally slow, but steady, and is very liable to produce definite symptoms of anæmia. The only treatment in such cases is to explore the uterine cavity manually and to remove the clot and plug the uterus if necessary. The author emphasizes the importance of such abnormal coagula formation in the ætiology of puerperal sepsis in cases which may appear to be normal.

Eclampsia.

W. J. DIECKMANN (*American Journal of Obstetrics and Gynecology*, September, 1931) advocates the use of 500 to 600 cubic centimetres of a 6% gum acacia solution in the treatment of eclampsia. Evidence based on blood volume, hæmoglobin, cell volume and serum protein estimations has been accumulated to prove that there is either an absolute or a relative decrease in blood volume in eclampsia. By using a gum solution this volume may be more satisfactorily restored than by other means. In the author's hands favourable results were obtained. In some cases the intravenous administration of glucose was also used.

Blood Extravasation and Albuminuria.

O'DONEL BROWNE (*The Journal of Obstetrics and Gynæcology of the British Empire*, Autumn Number, 1931), whilst agreeing with the generally accepted theory that nephritis can produce certain retroplacental changes which precipitate an attack of accidental hæmorrhage, emphasizes the fact that hæmorrhage alone will produce albuminuria. During his term of office as Assistant Master at the Rotunda Hospital, he tested, every two hours, the urine of patients with hæmorrhage and found that when the blood was retained albumin was present in the urine. This albuminuria disappeared some hours after the removal of the retained blood. Included in the series were cases of a hæmatoma of the vulva following a fall, ruptured ectopic pregnancy, and pelvic hæmatocele, in addition to the typical cases of accidental hæmorrhage. The albumin disappeared within a few hours to a few days of the removal of the clot. The author is emphatic that the urine must be tested frequently in order not to miss what may be only a transient phenomenon.

Labour with Congenital Dislocation of the Hips.

F. SCHRENK (*Deutsche Medizinische Wochenschrift*, December 18, 1931), after discussing the various theories regarding the ætiology of bilateral congenital dislocation of the hips, describes the course of labour in such a case. The lesion was diagnosed both clinically and radiologically. The femora lay just below the iliac crests, the whole pelvis was shallow and the inlet somewhat obliquely distorted. The mechanism of labour was that described as characteristic of flat pelvis, and once the head had passed the inlet, subsequent delivery was easy and quick; the second stage lasted fifteen minutes with a *primipara*.

The Use of Pituitary Extract in Labour.

H. KÜSTNER (*Deutsche Medizinische Wochenschrift*, January 1, 1932) describes a fatal case of uterine rupture following repeated doses of pituitary extract during labour. Taking this misuse of the drug as a text, he summarizes the indications for the use of stimulant drugs during the various stages of labour. For the induction of labour at term he prefers 0·5 cubic centimetre doses of thymophysin, repeated if necessary. Even in such small doses untoward results sometimes occur. Pituitary extract alone is very uncertain in its action. During the first stage drug stimulation is not only generally unnecessary, but often harmful. Morphine is a far more suitable drug, especially if the delay be due to spasm of the os. The use of pituitary extract is often recommended in the second stage, but he warns that it is very dangerous in the presence of any pelvic contraction. He uses it only in small doses, when the head is on the perineum. Its use is also forbidden if rapid delivery be essential, as in fœtal asphyxia. Better than pituitrin for most cases is chloroform or ethyl chloride. In the third stage he prefers to give pituitary extract, 0·5 to 0·75 cubic centimetre intravenously, as the subcutaneous route is too uncertain. During the puerperium small doses of ergot are sufficient, except in lochial retention, when intravenous use of pituitary extract is substituted.

Special Articles on Aids to Diagnosis.

(Contributed by Request.)

XV.

THE RADIOLOGICAL EXAMINATION OF THE UPPER PART OF THE ALIMENTARY TRACT.

Introduction.

On account of the barium meal examination being so frequently employed in modern medicine, an attempt has been made in the following article to outline for the benefit of general practitioners the principles underlying the method. It has not been considered necessary to enter freely into details of technique or the refinements of diagnosis which belong, of course, to the province of the radiological specialist.

The investigation of the alimentary tract by means of X rays is based on the fluoroscopic examination, or radioscopy, and the taking of films, or radiography, after the introduction of opaque material into the digestive tube. This material may be administered either in the form of a meal or a clysma. The opaque substance is barium sulphate, which must be of a high degree of purity, being specially prepared for administration in the large quantities used in these X ray procedures. It is suspended in various substances, usually carbohydrates, or in the form of mucilaginous emulsions. The essential is that the mixture should maintain the barium in suspension and not allow of rapid subsidence. In the case of opaque meals, palatability is an obvious desideratum. Many excellent preparations, with all the ingredients ready mixed and needing only the addition of water, are on the market.

Different procedures are adopted by different radiologists in regard to the administration of meals. In the single meal method the patient presents himself in the morning, having eaten nothing since going to bed the previous night. The opaque meal is swallowed, during and after which process the œsophagus and stomach are investigated. He returns four to six hours later and is examined in order to determine the rate of gastric emptying. In the double meal method the patient takes at home a bariumized carbohydrate meal four to six hours before coming in for examination. The patient in these circumstances usually attends at about midday or early in the afternoon, when the progress of the meal is noted and a second meal then given. The advantage of this method is that the patient is saved a visit and does not have to abstain from food for quite as long. On the other hand is the difficulty of getting the barium and instructions to the patient.

Provision by the clinician of an adequate history, with biochemical and other findings, where practicable, has a definite value in more ways than one. The time of the radiologist is saved and the patient is spared unnecessary visits and inconvenience if these data are made available before the X ray examination commences.

The relative values of fluoroscopy and radiography in investigation of the alimentary tract, especially of the stomach and duodenum, have been the subject of controversy in the past. The truth is that the two methods are complementary. With the stomach and duodenum especially, skilful palpation under the fluoroscope by an expert radiologist is the very essence of the examination. Radioscopic examination should never be rushed through in a few minutes. To exclude small lesions from every segment of the stomach, careful screening in all positions, from the erect to the Trendelenburg, is necessary. Films, properly taken, confirm the fluoroscopic findings and form a permanent and portable record to illustrate the findings for the physician or surgeon and also to serve as a basis for future comparison. Good serial films in the right and the left oblique positions are particularly helpful in outlining the pyloro-duodenal region and have robbed this area of many of its uncertainties. Powerful apparatus is essential to obtain satisfactory films in the short exposures necessary to prevent peristaltic blurring of the gastric outline.

In the earlier stages of the development of gastro-intestinal radiology much attention was paid to the so-called "indirect" signs of organic lesions of the stomach or duodenum, such as a six-hour gastric residue, hypermotility and hyperperistalsis. Nowadays, however, these signs serve their most valuable function in warning us that organic pathological change may be present and in intensifying our search for the direct evidence, the modern diagnosis being based on the "direct" demonstration of the actual lesion itself. A six-hourly residue is always suggestive of gastric cancer, gastric ulcer or duodenal ulcer. Sometimes it is a reflex manifestation of a pathological appendix or gall-bladder; and rarely is purely functional, unassociated with an organic lesion.

Œsophagus.

X ray examination of the œsophagus is a valuable preliminary to œsophagoscopy and may yield sufficient information to make this procedure unnecessary. The œsophagus is examined fluoroscopically during the swallowing of thin and thick barium boluses and barium-filled capsules, direct and oblique views, both in the erect and recumbent positions being employed. Normally the bolus passes quickly down the viscus, and so for the demonstration of intrinsic pathological change some degree of obstruction, either organic or spasmodic, is necessary. It has to be remembered that temporary arrest of a capsule or bolus may take place at the points of physiological narrowing, namely, at the levels of the cricoid cartilage, the suprasternal notch, the arch of the aorta, the left bronchus and the cardiac orifice. If a capsule sticks, it is suggestive of obstruction, and if not washed down after a swallow of water, obstruction by disease or a foreign body may be suspected.

Œsophageal Diverticula.

The diverticulum most commonly described is a congenital one at the upper end of the œsophagus; but it is not œsophageal and not wholly congenital, the symptoms appearing generally in men past middle age, and the condition originating as a herniation of the hypopharyngeal mucosa in the interval between the circular and oblique fibres of the inferior constrictor muscle of the pharynx. On X ray examination the barium collects in a rounded sac in the neck, and after the sac is filled, the barium passes down the œsophagus which lies in front of the sac. If this is large, the lower pole, filled with barium and showing a horizontal fluid level in the erect posture, may occupy the thorax behind the superior mediastinum. The large sacs do not completely fill with barium; they contain mucus and food, through which the heavier barium sinks to the lower pole. If sacs are entirely filled with food, no barium will enter and they remain undemonstrated. Congenital diverticula of the lower end of the œsophagus are very rare. Traction diverticula resulting from contraction of adhesions between the œsophagus and other thoracic structures, generally in the middle third, are also rare; they are often multiple, are small and cone-shaped, with the base of the cone opening into the œsophageal lumen. In the erect posture they may not fill.

Œsophageal Carcinoma.

In patients of the cancer age, even if a history of dysphagia is absent, it is wise to examine the œsophagus in the routine barium meal examination. Cases of suspected gastric carcinoma may be revealed to be œsophageal in site. Such a lesion may remain latent without any history of difficulty in swallowing, the dysphagia appearing quite suddenly, probably the result of inflammation and spasm developing around the edges of a hitherto symptomless malignant ulceration of the œsophageal wall. This type of lesion may be missed in the early stages during examination with the fluid opaque meal and even with a thick bolus. Its detection is facilitated by examination of the patient in the horizontal position, but probably the safest precaution is to administer a barium capsule. This will frequently, but not always, be arrested at the

site of an organic lesion. Many an experienced radiologist has felt the chagrin of having reported the œsophagus as normal and in a few weeks' time the patient returning with unequivocal evidence of carcinoma.

The ease of X ray demonstration of malignant disease, which appears to occur with equal frequency in the upper middle and lower thirds, depends on the type of growth. The contracting scirrhous and the cauliflower fungating types will produce early X ray evidence of obstruction, while the not infrequent area of ulceration projecting but little above the surface may yield no evidence until spasm develops. Œsophagoscopy should not therefore be delayed in a patient over middle age with symptoms referable to the œsophagus, in whom the X ray examination reveals no abnormality. X ray examination in carcinoma of the upper end of the œsophagus is difficult and unsatisfactory on account of the distress produced. The X ray characteristics of œsophageal carcinoma are a tortuous constriction with an irregular filling defect in the lumen above the stricture. The irregular indentations distinguish malignant disease from fibrous or spasmodic strictures, in which the margins of the retention shadow are smooth and rounded.

Benign stricture is not often seen, and there is generally a history of swallowing a corrosive fluid. All benign strictures, however, are not due to corrosive fluids. They may be due to tuberculosis, syphilis, prolonged sojourn of a foreign body, or trauma following instrumentation. Non-caustic fibrous stenosis occurring late in life suggests syphilis. The most common site of benign stricture is the middle third. Caustic strictures are often multiple and their lumina eccentrically placed. The majority of adult strictures are, of course, malignant.

Œsophageal carcinoma may produce a tracheo-bronchial fistula in which the bronchial tree is outlined with barium. Curiously enough, this complication is not invariably followed by a fatal septic pneumonia. A similar striking appearance may be produced in bulbar paralysis, where, with paralysis of the laryngeal and pharyngeal muscles, the barium runs directly into the trachea.

Cardiospasm.

One feels that with increased knowledge of the pathology of cardiospasm neither the term cardiospasm nor achalasia will be wholly acceptable. Radiological examination shows definitely that the stenosis is not at the cardia, but at the œsophageal opening in the diaphragm. Chevalier Jackson's view that this extensive dilatation of the thoracic œsophagus is due to a dysfunction of the normal "pinch-cock" mechanism of the diaphragm is more acceptable radiologically than the opinion that it is a spasm or non-relaxation of the cardiac sphincter. On the screen, after the barium has been swallowed, the thoracic œsophagus is seen to be greatly dilated, and terminates at the diaphragmatic aperture in the shape of a smooth, conical funnel, provided that the sac is not occupied by heavy, non-displaceable food fragments. If these are present, the conical termination may show filling defects, but the extreme dilatation of cardiospasm never obtains with carcinoma. The retention shadow may persist for hours. A definite hydrostatic pressure may overcome the obstruction, so that, with the patient taking repeated drinks, the upper level of the fluid column is seen to remain approximately constant in position. Sometimes a warm drink will temporarily relax the spasm.

Foreign Bodies.

The most common site for an œsophageal foreign body to lodge is the level of the suprasternal notch.

Opaque foreign bodies such as coins or safety pins are readily recognized on the screen; perhaps it is wise to confirm with barium swallows that they are in the œsophagus and not in the air passages. Coins in the œsophagus are found with the greatest diameter in the coronal plane, tracheal foreign bodies with their greatest diameter in the sagittal plane.

In the case of non-opaque and semi-opaque foreign bodies such as food boluses, fish bones, meat bones *et cetera*, a useful procedure is to give the patient small pledgets of cotton wool soaked in barium and water to swallow. These catch, in the majority of cases, on the projecting foreign body, the resultant skiagram demonstrating them hanging therefrom. Incidentally, their pearly white cotton wool attachment forms a guide for œsophagoscopic removal. A barium-filled capsule will usually, but not aways, be arrested at the site of a foreign body. Sometimes, when the screen gives no evidence, a foreign body may be demonstrated by films taken in various positions.

In some cases, after the onward passage of the object, patients may complain of a subjective sensation which suggests to them that the foreign body is still present in the œsophagus. It is not always wise to assume because the X ray examination fails to reveal a foreign body that none is present. In such cases œsophagoscopy is, of course, the counsel of perfection.

Spasms Other Than Cardiospasm.

Spasms of the œsophagus other than cardiospasm, sufficient to cause intermittent dysphagia of more or less severity, may be due to intrinsic œsophageal conditions or be reflex from diseases of the stomach, duodenum or gall-bladder, or be purely functional. The latter two types may temporarily arrest a capsule, but a long drink of water will generally pass it on. In those spasms associated with intrinsic œsophageal lesions the capsule will remain *in situ*. There is a type of spasm affecting the crico-pharyngeus muscle in which the difficulty is in starting the bolus downwards. Some forms of *globus hystericus* belong to this category. In organic nervous diseases, for example, bulbar palsy, affecting the œsophagus, the bolus will not descend. While intrinsic œsophageal disease generally causes spasm near its site, spasm of the inferior pharyngeal constrictor may result from lesions lower down. Sometimes the only indication of an early carcinoma is spasm, and this is true of superficial, peptic, tuberculous and syphilitic ulcer, all of which are, of course, rare. Peptic ulcer occurring at the lower end of the œsophagus and producing symptoms not unlike those of gastric or of duodenal ulcer, may be superficial, when it can only be suspected by the spasm induced, or deep, when a projecting niche is evident.

Stomach.

The stomach is the most important, difficult, and withal, fascinating section, and the one giving most scope for skilful and intelligent development of radiological technique. The importance of a searching examination and correct deductions therefrom is due to the high incidence of both carcinoma and peptic ulcer. As the late Russell Carman pointed out, the requisites for efficient examination of the stomach include a knowledge of the morbid anatomy of the stomach, a technique designed to reveal pathological conditions, whatever their character or situation may be, a knowledge of the normal stomach, and a thorough acquaintance with the protean manifestations of gastro-spasm. The many difficulties of barium meal examination are greatly increased with stout patients: radioscopy takes longer, palpation is more difficult, more films with longer exposures are required, and lesions cannot be so clearly recorded as in thin subjects. Complete eye accommodation before commencing screening is, of course, essential in all cases. From the older method of administering the whole opaque meal to the patient and then examining the filled stomach, has developed the method of following one or two mouthfuls of the barium suspension in its course into and along the viscus. This small quantity of opaque material is manipulated so as to smear or "whitewash" the various portions of the mucosal surface, which can thus be examined in detail. The irregular pattern of the mucosal folds or rugæ of the gastric mucosa, however, makes such observations and the corresponding deductions anything but easy. A careful examination of the onward progress of the head of the first mouthful should be made, especially at the cardiac end of the stomach, immediately after its entrance. The first indication of a carcinoma in this area may be the splitting of the contrast material into two paths on meeting a malignant mass.

Gastric Ulcers.

The incidence, as shown radiologically, of gastric ulcer, is very much less than duodenal ulcer, and the ease of X ray demonstration of gastric ulcer depends on its nature, whether acute or chronic, penetrating or non-penetrating, innocent or malignant, and on its position on the gastric walls. The classical niche projecting from the lesser curvature of the *pars media*, with an associated notch on the greater curvature, is too well known to be described here; unfortunately all gastric ulcers are not so obvious and may be entirely missed in the hands of the inexperienced. A small superficial ulcer, especially on the posterior wall, may not retain any of the barium emulsion. Sometimes the ability to demonstrate and reproduce a small spot or "fleck" by smearing the barium suspension with the fingers over the involved area may be the only clue. Some convergence of the rugæ is occasionally demonstrable in films taken with the area under compression. The apparent depth of peptic ulcers is often exaggerated by swelling of the surrounding mucous membrane, subsidence of the latter suggesting erroneously at a later examination a great diminution in depth of the real ulcer crater. Demonstration of a niche does not therefore indicate a penetrating ulcer: it may be entirely intramural.

One should call attention to the great frequency with which no lesion can be demonstrated following a recent hæmatemesis, the latter in these cases originating from a small area of superficial erosion or ulceration and with no swelling of the surrounding mucosa, owing to the effect of the blood-letting. Sometimes the presence of an ulcer may be obscured by the presence of blood clot or food *débris* in the crater. In the latter case, reexamination within a day or two may demonstrate a definite crater. The same source of error may obtain when the healing of an ulcer is in question. In passing, it might be stated that there is convincing X ray evidence that even large ulcers may heal (at least temporarily) under medical treatment, as shown by the complete disappearance of the crater. Healing ulcers frequently show a conical niche. A warning must be issued against too vigorous palpation where there is much local tenderness, as the possibility of rupturing an ulcer is in such a case very real. Where a duodenal ulcer is suspected and nothing abnormal detected in that area, it is well to make the examination with particular care for a gastric ulcer fairly high up in the *pars media*, as the symptoms of these two conditions may be surprisingly similar. Retraction of the prepyloric segment on the lesser curvature side is also frequently an indication of the presence of an ulcer higher up on the lesser curvature. An incisura of the greater curvature, due to spasmodic contraction of the circular muscle fibres involved in the ulcer, is also an obvious indicator. This "indirect" sign, however, is frequently entirely absent, especially with the smaller, more superficial ulcers. In the case of chronic ulcers, cicatrization, frequently with an associated spasmodic factor, may so constrict the involved zone as to produce the well known bilocular or "hour-glass" stomach. It is not possible to decide preoperatively how much of the biloculation is due to spasm and how much to cicatrix. Large ulcer craters are open to suspicion, for those over 2·5 centimetres in diameter are often malignant. It should be borne in mind that in 10% of gastric ulcers an associated duodenal ulcer is also present.

Prepyloric ulcers, which constitute about 10% of gastric ulcers, are the most difficult to detect, and the differential diagnosis includes carcinoma, innocent tumours, syphilis, gastrospasm, hypertrophy of the pyloric musculature and extrinsic conditions, such as adhesions. Prepyloric pathological change can be extremely puzzling; suffice it to say that the typical ulcer niche is only rarely seen in this area, and the radiologist's abilities are frequently greatly taxed in arriving at a conclusive diagnosis. Often this cannot be given.

Rupture of a hollow viscus, for example, perforation of a peptic ulcer, may sometimes be demonstrated by the presence of gas under the right dome of the diaphragm. This is of diagnostic value in the differential diagnosis of an "acute abdomen".

Gastritis.

Efforts are being made to extend the study of the pattern of the gastric rugæ to the diagnosis of the atrophic, hypertrophic and granular types of gastritis, and some Continental authorities have gone so far as to describe certain forms of gastritis preceding ulcer and carcinoma; but with the limits of the normal rugal appearances not yet fully defined, one feels that the sources of error are too great for reliance to be placed at present on this refinement of X ray diagnosis.

Gastric Carcinoma.

The appearances with carcinoma depend on whether the neoplasm is of the scirrhous, fungating or ulcerative variety, the essential features being a persistently irregular filling defect, often with a palpable mass. The radiologist's method of examining the patient in the erect posture accounts for the ease with which the mass can so often be detected—a point sometimes not fully appreciated by the clinician, who frequently carries out his investigations with the patient supine on the examining couch.

While the filling defect is the basic Röntgen sign of carcinoma, other conditions may produce filling defects; we have often to invoke the aid of additional radiological signs before arriving at a diagnosis of carcinoma. Of these other signs, one is the absence of peristalsis from the involved area. Flexibility and mobility are impaired and there is often an alteration in size; shrinking is the rule, dilatation being only rarely seen. Motility, as shown by the six-hourly observation, may be expressed as: (i) a rapid emptying due to a "gaping" infiltrated pylorus or to the associated achlorhydria, or (ii) a delay due to pyloric obstruction. The presence of a palpable tumour corresponding to a defective appearance is, of course, highly suspicious. Though "ulcer-tumours" do occur, a tumour will usually prove to be a carcinoma.

Manipulation of the partly filled stomach is of great importance. Disappearance of the rugæ at the boundaries of the carcinomatous area is a valuable diagnostic point; carcinomatous masses on the posterior wall, and the curvatures uninvolved, may escape detection except with pressure, the full stomach presenting an entirely normal appearance. Even where the latter shows a definite filling defect, as with a fungating prepyloric carcinoma, this compression manœuvre will demonstrate more effectively the limits of the lesion. Pseudo filling defects may be caused by extrinsic pressure from tumours in the liver, spleen, pancreas, kidney or mesentery. On the greater curvature side pressure from excessive gas in the splenic flexure is a trap for the unwary. On the lesser curvature a pancreatic cyst produces a characteristic picture. Unless the tumours are adherent to the stomach, palpation and alteration in appearance, with change of position, will establish their extragastric origin.

In considering resectability it is important to remember that infiltration is generally more extensive than is shown on the screen or film. Owing to the symptom-free period of gastric carcinoma, the great majority are well advanced by the time the patient comes for X ray examination. Here, as in the X ray examination of other systems, it is easier to give a positive than a negative diagnosis, as an early growth may be impossible to diagnose. If in doubt, the radiologist should not hesitate to ask for a reexamination in some weeks' time.

Innocent Tumours.

Innocent tumours—myomata, adenomata, polypi—are very rare. They are demonstrated as rounded defects, usually on the posterior wall, with the rugæ in the sessile variety stopping abruptly at the filling defect. A further diagnostic point is that the curvatures as a rule are not involved.

The polypoid type are usually prepyloric and sometimes prolapse through the pylorus into the duodenum. Their tendency to undergo malignant change has to be borne in mind. The majority of these benign tumours are soft and compressible and generally a defined mass corres-

ponding to the defect is not palpable. Multiple gastric polyposis gives a characteristic X ray appearance.

Gastric Syphilis.

Gastric syphilis usually shows as a concentric filling defect involving both curvatures; there is no palpable mass; the involved portion of the stomach is more flexible than in carcinoma, whilst the general condition of the patient is much better than would be the case with so extensive a malignant disease.

Diverticula.

Diverticula of the stomach occur most frequently near the œsophageal entrance, but may also occur in the middle third projecting from the lesser curvature, when they closely simulate a penetrating ulcer. The smoothness of the edges, the presence of a communicating stalk and the absence of associated spasm and cicatrix help in their recognition.

The Post-Operative Stomach.

Examination of the stomach after gastro-enterostomy or partial gastrectomy on account of return of symptoms often provides food for thought for the radiologist in as much as he may fail to demonstrate any organic cause for the return of the dyspepsia. In some gastro-enterostomy cases there may be a small six-hourly residue lying between the stoma and the pylorus. If the pyloric route is open, a residue suggests a pyloric spasm occurring at the end of digestion, most probably caused by a return of the duodenal ulcer for which the operation was performed. Again, there may be evidence of dilatation of the efferent loop, due to the non-establishment of sphincteric control at a gaping stoma. Kinking or excessive adhesions may obstruct the jejunum. It is not unusual for these jejunal obstructions to be intermittent, depending probably on the degree of gastric distension. The direct demonstration of jejunal ulcer is very difficult, owing both to the shallowness of the ulcer crater and to the general obscurity produced by overlapping barium shadows; "indirect" signs have often to be relied on and a diagnosis of jejunal ulcer cannot be easily made even by the skilled radiologist.

Duodenum.

The Normal Duodenum.

The most important part of the duodenum from the radiologist's viewpoint is the duodenal cap, which is the first inch of the superior part. Unlike the remainder of the duodenum, it possesses no *valvulæ conniventes*, therefore, when it is filled, a homogeneous shadow results. Rugæ similar to those in the stomach may be demonstrated, that is, longitudinal rugæ along the lesser curvature and tortuous rugæ along the greater curvature. Its normal shape may be triangular, circular or elliptical, and its position also varies, the shape, position and size depending largely on the habitus of the patient. In hypersthenic patients the pyloric canal is often directed backwards, and the duodenal cap lies partly behind the pyloric antrum; this makes examination of the cap in obese, hypersthenic patients a difficult procedure; in thin patients, on the other hand, the cap can be much more easily and conveniently studied. The remainder of the duodenum possesses *valvulæ conniventes*; therefore, filling is irregular and non-homogeneous.

Technique of Examination.

The technique is, as elsewhere in the alimentary tract, a combination both of the radioscopic and of the radiographic. Palpation during the screening is essential, and is designed to distend, as far as it is distensible, the cap, and to keep it distended during part of its examination. This is of great importance, as it is on the appearance of the barium distended cap that the diagnosis of pathological change is most frequently made. If no palpation is used, the normal cap may not fill completely, and false deductions may be drawn therefrom. Palpatory compression of the anterior and posterior walls must not be omitted, and is equally as important as the palpatory distension.

In order to view all the peripheral surface of the cap, it is necessary to examine in many positions, of which the right and the left oblique are usually the most important. Screening by itself of the duodenal area is often insufficient, and in addition to providing a permanent record, a properly exposed series of films may reveal abnormalities which were not detectable by the naked eye. This is no reflection on the ability or the care of the observer, but simply a question of the limits of human visual acuity. A series of films is necessary, as a single film may be most misleading. One of the latest developments of gastro-intestinal X ray equipment is a device for taking "snapshots" of the cap under compression.

Duodenal Ulcer.

Duodenal ulcer is the most common lesion of the upper alimentary tract, being six times as common as gastric ulcer. The first local radiological sign—"the persistent bulbar fleck"—was described in 1910 by Arthur Barclay, now Lecturer in Radiology at Cambridge, and a high standard of precision and accuracy in the X ray diagnosis has since been developed. Åkerlund analysed varying cap deformities produced by ulcer, and showed the presence, either singly or in combination, of the following signs, namely, "niche" or "fleck" formation, characteristic spasm on the curvatures, cicatricial contractions, pseudo-diverticulum and eccentric placing of the pylorus in relation to the base of the cap. Åkerlund's masterly work still remains the sheet anchor of diagnosis. The typical Åkerlund deformities are produced by nothing else than ulcer, though other deformities can be produced by extrinsic conditions, varying from enlarged gall-bladder to ureteric calculi. The great majority of duodenal ulcers are limited to the cap. The ulcer crater may be a niche or a fleck and the usual shallowness of the duodenal ulcer crater explains why this sign is lacking so frequently, though modern methods of technique are greatly increasing the number of cases in which a niche is demonstrated.

Duodenal ulcers are situated with equal frequency on the anterior and posterior walls, but the curvatures *per se* are rarely involved. This is the reason why a single postero-anterior view may leave the ulcer crater entirely unrevealed. The fleck or tiny residue in a small crater is elicited by pressing hard on the distended cap and so approximating the anterior and posterior walls. It is then known as the *niche en face*. A *niche en face* in one view may be a niche in profile in another. The *niche en face* may be surrounded by a circular clear zone, caused by mucosal swelling around the ulcer periphery. The presence of a niche is not essential for diagnosis unless no other deformity exists. The niche is less common in the distal half of the bulb, and lies more to the lesser curvature than to the greater curvature side. Sometimes two ulcer niches are seen placed opposite each other (Moynihan's "kissing ulcers").

In the absence of an ulcer niche we must, in the majority of cases, rely on the other elements of cap deformity. While spasm in the stomach is a hindrance, in the duodenal cap it is a very definite aid in the detection of ulcer. The most characteristic spasm is a deep one on the greater curvature side, and is similar to the incisura of the stomach. Retraction of the lesser curvature due to spasm of the longitudinal fibres is also commonly shown, and this produces the eccentric placing of the pylorus. A localized dilatation of the cap proximal to a persistent spasm or cicatrix not infrequently occurs at the junction of the base and the greater curvature, producing a "pseudo-diverticulum" (Moynihan's "pouched ulcer"). It is not always possible to decide whether cap defects are due to spasms or cicatrices. Sometimes these defects encroach on the pylorus, and even on the stomach, and it becomes difficult to tell whether the ulcer is prepyloric or postpyloric. In the majority of these doubtful cases the lesion is postpyloric.

These spasms and the cicatrices are responsible for the various well known appearances of the cap known as "clover-leaf", "branch-of-coral", "pine-tree", "oak-leaf", which, when persistent, are characteristic of ulcer. Spastic deformities seem to be less common when the niche is centrally situated, and the latter is likely to be overlooked

unless the cap is examined and manipulated and compressed in numerous positions.

The indirect signs of duodenal ulcer have, like all indirect signs, a limited value in diagnosis. They are gastric hyperperistalsis, hypermotility (as shown by the head of the meal having advanced to the distal colon after six hours), and gastric residue after six hours. In obstructive ulcer the cause of the gastric stasis is obvious; in non-obstructive ulcers it is often only a tiny residue, and probably due to pyloric spasm occurring at the end of digestion.

The aid of the radiologist is often invoked as to whether or not a duodenal ulcer has healed, and only rarely can he answer this question with assurance. In those cases where an ulcer crater has been convincingly demonstrated, its disappearance after treatment may be shown, but where only the characteristic spastic cap deformities enabled the clinical diagnosis to be confirmed originally, the problem is distinctly different. Just in the same way as a gastric incisura may persist after healing of a crater on the lesser curvature of the stomach, so the various cap spasms (and, of course, cicatrices) may persist after a duodenal ulcer has healed. This phenomenon has led to the dictum, "once a duodenal ulcer radiologically, always a duodenal ulcer". The test for occult blood in the stools is more informative than X ray examination.

A tiny duodenal ulcer may be present without producing any cap deformity; the crater is too small and too shallow to produce a niche, and the cap may show no spasm. It is therefore not uncommon in cases of hæmatemesis, due to a small acute ulcer, for the X ray examination to reveal no abnormality.

The cap deformities due to ulcer have to be distinguished from spasm caused reflexly by extrinsic causes such as cholecystitis, appendicitis *et cetera*. In such cases the deformity is not persistent; it does not conform to Åkerlund's criteria; it can be relieved by atropine; and there is no tenderness on palpation directly over the cap. It might be mentioned that local tenderness over a duodenal ulcer may be entirely absent.

Periduodenal adhesions may cause deformities indistinguishable from ulcer. The diagnosis of adhesions should not be lightly made. It is only rarely that they deform the cap, while, on the other hand, they may exist without distorting the duodenal outline. Any suggestion of pericholecystitis warrants a cholecystography.

Pressure from an enlarged gall-bladder may produce a cap deformity, usually manifested as a crescentic filling defect on the greater curvature. This is one of the "indirect" signs of pathological gall-bladder, but is not always reliable (a normal gall-bladder as well as a lobe of the liver may give such an impression on the cap outline). Fortunately, with the advent of cholecystography we have a much more valuable method of diagnosing pathological change in the gall-bladder.

Duodenal Diverticula.

Congenital duodenal diverticula are not uncommon; their common site is around the duodenal papilla and the duodeno-jejunal flexure, and they project into the curve of the duodenum. They practically never occur in the cap. Whether or not they are filled with barium depends on the presence or absence of food in them at the time of examination, and their size varies from that of threepence to that of a florin or larger. Their outline is sharp and well defined, and their filling homogeneous, due to the absence of *valvulæ conniventes*. The channel connecting the diverticulum in the majority of cases is absent. There may be considerable delay in these cavities. Practically never are they to be confused with duodenal ulcers.

Duodenal Tumours.

Duodenal carcinoma is certainly very rare, but it does occur and should be borne in mind as a cause of duodenal obstruction and dilatation; its site is usually at the junction of the first and second portions or in the vicinity of the ampulla of Vater. Benign tumours, also very rare, are usually polypi, and they cause a rounded, well defined defect in the cap and produce a gastric retention.

Duodenal Ileus.

Chronic duodenal obstruction may be due to compression by the superior mesenteric artery in gross enteroptosis, the obstruction affecting the third part in the mid-line. The whole of the duodenum proximal to the compression is seen to be dilated, and duodenal stasis, with the stomach empty, may occur for considerably after six hours. The ileus is often intermittent and so may not be evident at a single examination. Other cases are due to constriction by the middle colic artery, by periduodenal adhesions, and by adjacent tumours, for example, carcinoma of the pancreas. Considerable dilatation of the cap alone is not infrequently revealed, unassociated with any obstruction or demonstrable lesion. It occurs in the prolapsed hypotonic stomach, and the cap may attain a very large size, though it does not appear to have any great clinical significance.

Jejunum and Ileum.

The jejunal coils occupy the left side of the abdomen and are recognized by the snow-flaky, feathery outlines of the *valvulæ conniventes*; the ileal coils occupy the right side of the abdomen, and the pelvis and their barium contents show a dense, uniform shadow, due both to the absence of *valvulæ conniventes* and to the slower rate of passage along this portion of the bowel. The transition from jejunum to ileum is gradual and takes place in the mid-line about the third lumbar vertebra.

The rate of passage through the small intestine varies with the individual and, of course, depends on the emptying rate of the stomach. With the stomach empty in four to six hours, the small intestine is generally completely empty in twelve hours. Back pressure from colonic stasis may be responsible for ileal stasis. All the coils of small intestine should be freely mobile to palpation, but it is somewhat difficult to displace coils that are deeply situated in the pelvis in enteroptosis, and failure to achieve this displacement should be interpreted only with caution as due to fixation by adhesions.

The lesions affecting the small intestine are not common and are difficult to diagnose unless producing obstruction. Obstruction may sometimes be diagnosed fluoroscopically without an opaque meal by the formation of numerous fluid levels with gas above. The exact site of the obstruction may not be evident, but the diagnosis by means of plain films is naturally quicker than when a barium meal has to be given. The most frequent causes of chronic obstruction are ileo-caecal tuberculosis, adhesions and neoplasm. When viewed after barium meals, the obstructed small intestine shows the coils of bowel dilated and unfolded, and tending to be arranged vertically. Fluid levels with gas above are prominent. The feathery appearance of the jejunum has given place to a "concertina" pattern.

Adhesions or bands very often fail to produce any demonstrable X ray evidence, while, on the other hand, adhesions must not be diagnosed too readily. Angulation or "kinks" in the small bowel, unless accompanied by fixation, dilatation or retention, usually possess no clinical significance.

Diverticula of the small gut occur in two forms, the large pouch and the multiple type. In the multiple type diverticula are usually also found in the duodenum and colon. A Meckel's diverticulum can rarely be recognized, owing to the difficulty of distinguishing a small fluid loop amongst the closely packed ileal coils.

Malposition and displacements of the coils are quickly detected—pelvic tumours (and a distended bladder) displace the ileal coils upwards, tumours of the pancreas displace the jejunum downwards, those of the spleen towards the mid-line, and renal tumours downwards and towards the mid-line. Appendiceal and other pelvic abscesses cause loops of small bowel to encircle the tumour in an endeavour to wall it off. The loops around the appendiceal abscess are generally dilated, owing to a toxic ileus.

Conclusion.

It is impossible in a short article, such as this, to do anything more than touch very briefly on the more important aspects of X ray examination of the upper part of the alimentary tract.

It is generally agreed that the X ray method of examination in skilled hands is the most important in the elucidation of gastro-duodenal pathological change, but the limitations of the method and the fruitful sources of error to the untrained must always be borne in mind.

On account of the importance of modern surgery in the treatment of conditions of the alimentary tract, deductions made by observers possessed of any but the best apparatus and ill-equipped with a knowledge of normal X ray appearances and of the pitfalls of spasm, may be nothing less than disastrous.

K. STUART CROSS, M.D., M.Sc.,
Lecturer in Radiology, The University of Melbourne.

British Medical Association News.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Long, William John, Junior, M.B., B.S., 1930 (Univ. Melbourne), Base Hospital, Bendigo.

O'Brien, Augustine Byrne, L.R.C.S., L.R.C.P., 1929 (Edinburgh), R.F.P.S., 1929 (Glasgow), Kerang.

Sewell, James Erskine, M.B., B.S., 1930 (Univ. Melbourne), Children's Hospital, Carlton, N.3.

Correspondence.

SECTION OF OTO-RHINO-LARYNGOLOGY: ANNUAL MEETING OF THE BRITISH MEDICAL ASSOCIATION.

SIR: By today's English mail I received a communication from Sir St. Clair Thomson, the President of the Section of Oto-Laryngology at the Centenary Meeting of the British Medical Association, London, 1932.

As President of the Section he is very anxious to have the names of any members of our specialty who intend being present at the meeting. I shall be pleased if you will give space for this letter in the pages of the journal. As it is desired to have the names of practitioners who are likely to be present at the meeting, as soon as possible, I suggest that any readers interested might communicate with me at once in order that I may forward the information without further delay.

Yours, etc.,

R. GRAHAM BROWN,
Vice-President,
Section of Oto-Rhino-Laryngology.

Preston House,
Queen Street, Brisbane,
March 22, 1932.

NASAL SINUSITIS.

SIR: I have read with much interest Dr. Clive Eadie's article in the February 20 issue of your journal, and I think he is to be congratulated, as it marks a distinct step in the advancement of *post mortem* examinations. It likewise brings into prominence the important part sinus infection is causing in disease. For long it has been evident that it was responsible for practically all the chronic bronchitis, bronchiectasis and a large number of asthmatics. In this infection, which is almost universal in our communities, it is uncommon to find one source of infection only, the tonsils being invariably involved and frequently the gums. It is more than probable that it all originates from the tonsils, which are infected by the parents at a very early age. More enlightenment might have been evolved if attention had also been given to these localities at the time of the *post mortem* examinations. In March, 1920, I drew attention to the number of infections in just on 1,000 cases at the Melbourne Hospital, giving the various infections in each individual. In 1925 attention was again drawn to the prevalence of sinus infections, where 25% of out-patients were found to be involved. As in the majority of cases the same organisms are to be found in each of the localities, but not invariably so, it is not to be wondered at that disappointments are frequently encountered where only partial removal is adopted. I would plead for the more thorough investigation in all cases of goitre, especially the exophthalmic type, as time and time again has the clearing up of infected sinuses brought about a cure. At the present stage of our knowledge we have to own that the treatment of sinus infection is far from satisfactory, and there is great scope for anyone, specialist or otherwise, who is gifted with a lively imagination, to try out any scheme which offers hope of success or even improvement. "Colloidal manganese" should always be given a trial, as in numbers of cases the results are quite dramatic. A combination of operation with ionization, after Friel's technique, is worthy of an exhaustive trial.

Yours, etc.,

SYDNEY PERN.

12, Collins Street,
Melbourne,
February 29, 1932.

DIATHERMY OF TONSILS.

SIR: Dr. Kent Hughes, in his letter of your issue of March 2, 1932, in reply to my question as to whether he had the courage to request his colleagues who had seen his unsatisfactory results of diathermy of tonsils, to bring them to light in these columns, asks what is behind my letter.

I would assure him of my singleness of purpose, namely, to convince him of his errors or to be myself convinced in the attempt. I have not seen any of his results, good or bad, nor have I any inside information on them.

On the other hand, I have seen the results of cases treated by others, and many of them are very different from those claimed by Dr. Kent Hughes. In fact, I cannot myself reconcile the results at all, even assuming that Dr. Kent Hughes has a wizard's skill and diathermy machines of unusual frequencies and shapes of wave. It is to give him a chance to prove he can produce results incomparably better than others that I asked my question.

I am disappointed that he did not follow my suggestion, but I do not think the less of him in consequence, as it would require very great courage to have done so, and unlimited confidence in the method. However, he has now asked all who have seen unfavourable results in his cases he has treated by diathermy of tonsils, to report them to Dr. Gutteridge. I trust he will further ask Dr. Gutteridge to summarize the reports and to make available a report on them in order to let one get a quite unbiased opinion. I hope Dr. Kent Hughes will not resent my suggestion that he is biased in favour of diathermy of tonsils, any more than I would if he suggested I was strongly biased against it.

Dr. Kent Hughes claims that diathermy of tonsils: (i) Is the best method of tonsillectomy. (ii) Can completely remove tonsils. (iii) If it fails to remove any portion, sterilizes what remains. (iv) Is painless. (v) Does not produce any post-operative hæmorrhage.

I claim that diathermy of tonsils: (i) Is a very poor second best method of tonsillectomy. (ii) Cannot completely remove the tonsil without extensive peritonsillar damage. The penetration cannot possibly be controlled to stop evenly at the capsule, a structure that is less than 0·5 millimetre thick. The size of the remnants that one can dissect out after the patient is told his tonsils have been completely removed by diathermy is astonishing. (iii) Does not sterilize the portions left and, if it did

that, the infection could return. Dr. Kent Hughes can verify this if he will do some experimental work, in conjunction with his pathologist. (iv) Sometimes is accompanied by scarcely any pain, but more often there is moderate or severe pain, which has to be repeated a large number of times, so that, in very many cases, the patient suffers as much or more in the long run than with a well performed dissection. (v) Is sometimes followed by alarming secondary hæmorrhage, which is much more difficult to control than reactionary hæmorrhage. (vi) Gives very little immunity from recurrence in the case of previous repeated quinsies, where there is a very thick adventitious capsule.

Strangely enough, practically every ear, nose and throat consultant with whom I have spoken on the subject, has come to corresponding conclusions.

Six or eight years ago there was a wave of diathermy of tonsils in Europe, but little routine diathermy of tonsils is done there now. Two years ago there was a similar wave in Australia, but the number of those who now consider it better than tonsillectomy is very small, and there is every indication of them becoming fewer.

Dr. Kent Hughes asks my reasons why I think tonsils should be removed completely. I reply that they should be removed completely for the same reason that they should be removed at all, and couple my remarks above in the paragraph marked (iii).

Trusting either I or Dr. Kent Hughes will convince the other.

Yours, etc.,

A. B. K. Watkins.

Newcastle,
New South Wales,
March 14, 1932.

Corrigendum.

Dr. J. Colvin Storey points out that certain figures were omitted from Table II in his article on acute abdominal conditions appearing in the issue of April 2, 1932.

The figures for perforated duodenal ulcer and diverticulitis for four years were omitted. The number of cases of perforated duodenal ulcer was 74, the deaths numbered 19 and the percentage death rate was 25·67. The figures for diverticulitis were 37, 2 and 5·94. The words "requiring operation" should have been omitted from the heading of Table II.

Diary for the Month.

Apr. 12.—New South Wales Branch, B.M.A.: Ethics Committee.
Apr. 14.—New South Wales Branch, B.M.A.: Clinical Meeting.
Apr. 19.—New South Wales Branch B.M.A.: Executive and Finance Committee.
Apr. 22.—Queensland Branch, B.M.A.: Council.
Apr. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Apr. 27.—Victorian Branch, B.M.A.: Council.
Apr. 28.—South Australian Branch, B.M.A.: Branch.
Apr. 28.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xiv.

Balmain and District Hospital, Sydney, New South Wales: Resident Medical Officer.

Sydney Hospital, Sydney, New South Wales: Honorary Officers.

The Brisbane and South Coast Hospitals Board, Queensland: Honorary Officers.

The Otago Hospital Board, Dunedin, New Zealand: Resident Medical Officer.

The Rachel Forster Hospital for Women and Children, Sydney, New South Wales: Honorary Clinical Assistant to Out-Patients (Female).

The University of Melbourne, Victoria: Acting Lectureship.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
South Australian: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

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